

# EXAMINATION OF CVS & murmur (in short )



By  
Dr . Shamol  
FCPS (medicine)

shamol  
DR SHAMOL

ALWAYS FOLLOW THE TEXT  
BED SIDE TEACHER IS YOUR BEST TEACHER IN CLINICAL EXAM  
IF YOU FEEL ANY –CONTRADICTION FOLLOW THE TEXT  
IF YOU FIND ANY ERROR PL INBOX ME  
GIVE THE CORRECTION IN COMMENT

It is consist of

Peripheral pulse

BP

JVP

Examination of precordium

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## Examination of precordium

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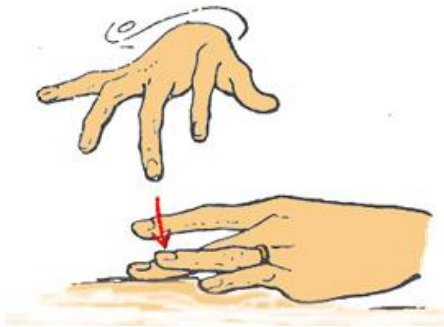
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INSPECTION

PALPATION



PERCUSSION

AUSCULTATION



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Inspection

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stand right side of the patient

As-Salam-u-Alaikum

Give Salam

Introduce yourself to the patient



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Now proper exposure  
Remove the clothes

Proper position of the  
patient –arm should be  
full abducted so that you  
see the lateral surface of  
the thorax



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## What to see ?

1. Size And Shape and any deformity of the Chest Wall ( **see in respiratory system pdf** )
2. Visible impulse
3. Scar mark, Engorged vein present or not ,  
pigmentation---hypo or hyper pigmentation or  
fungal infection ( **go to respiration system** )
4. Other ( **go to respiratory system PDF** )
  - a) Feature of respiratory disdistress
  - b) Neck swelling –SVO,
  - c) Gynaecomastia and spider nevi and  
pigmentation

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Visible impulse

Please look for visible pulsation

in apical area and in other area

**3 suprasternal and supraclavicular pulsation**

**1. Cardiac impulse &**  
Usually u may see visible impulse  
in precordium or apical region

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**2. epigastric pulsation**

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Please look for visible apex beat or apical impulse or other pulsation

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suprasternal  
and  
supraclavicular  
pulsation

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Please look for epigastric pulsation present or not

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PALPATION

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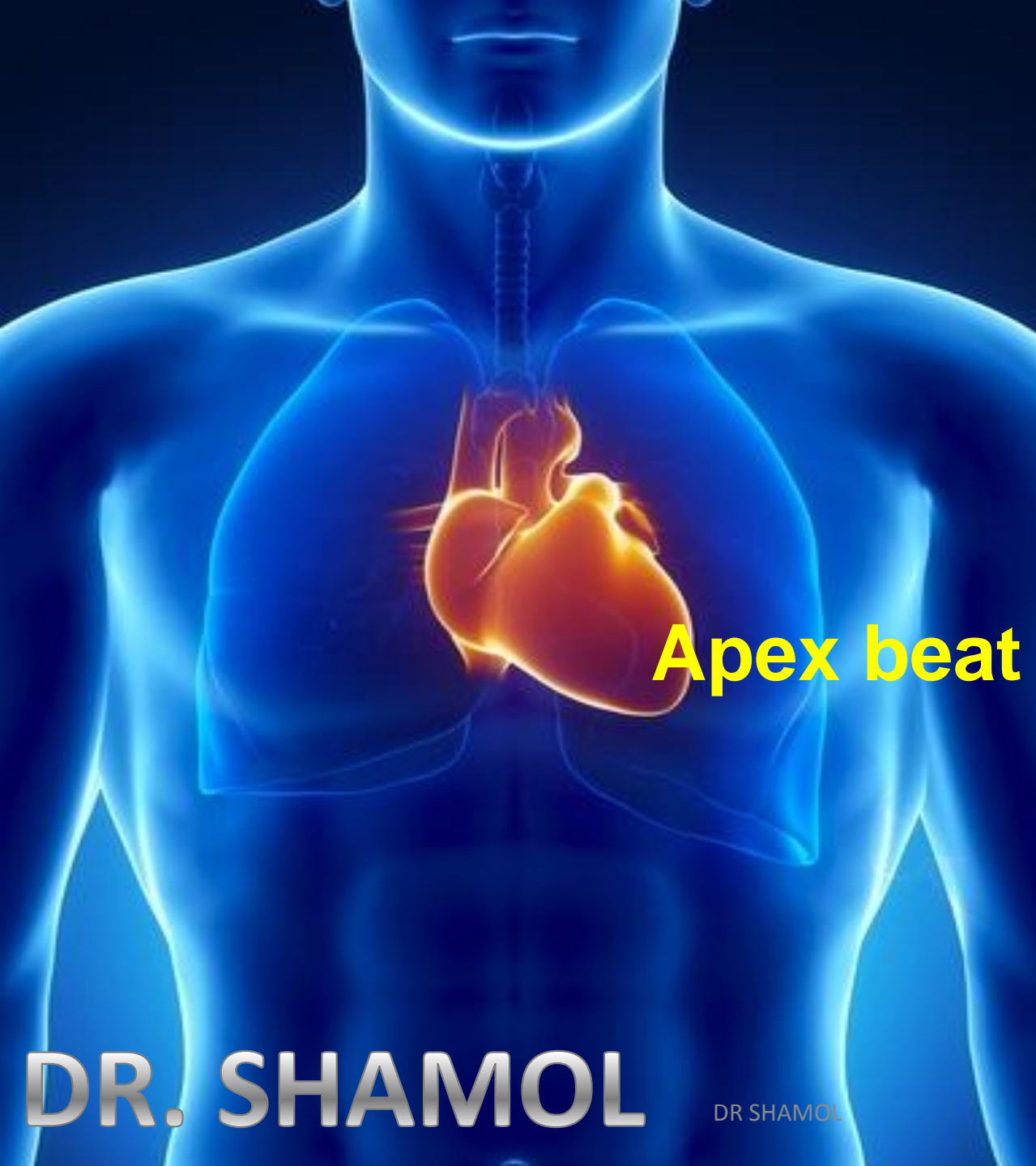
**In palpation we will  
see the following**

**Apex beat**

**Left parasternal heave**

**Palpable P<sub>2</sub>**

**Thrill**



**Apex beat**

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First keep the measuring tap on the bed from your pocket as it will need later to measure distance of apex beat from mid line

Place the flat surface (palm) of your right hand just below the nipple try to feel any cardiac pulsation present or not.

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It is better to place the hand more laterally

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Now move the  
hand toward  
medially to feel  
and locate the  
apex

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Npw if you feel the apex then localize the apex beat with single finger

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Either u got apex beat or not roll the patient left laterally

if u got the apex beat previously then it help u to confirm the apex or  
if can not locate the apex it help you to located the apex beat

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Now return the patient in his previous position and localize the apex beat with single finger.

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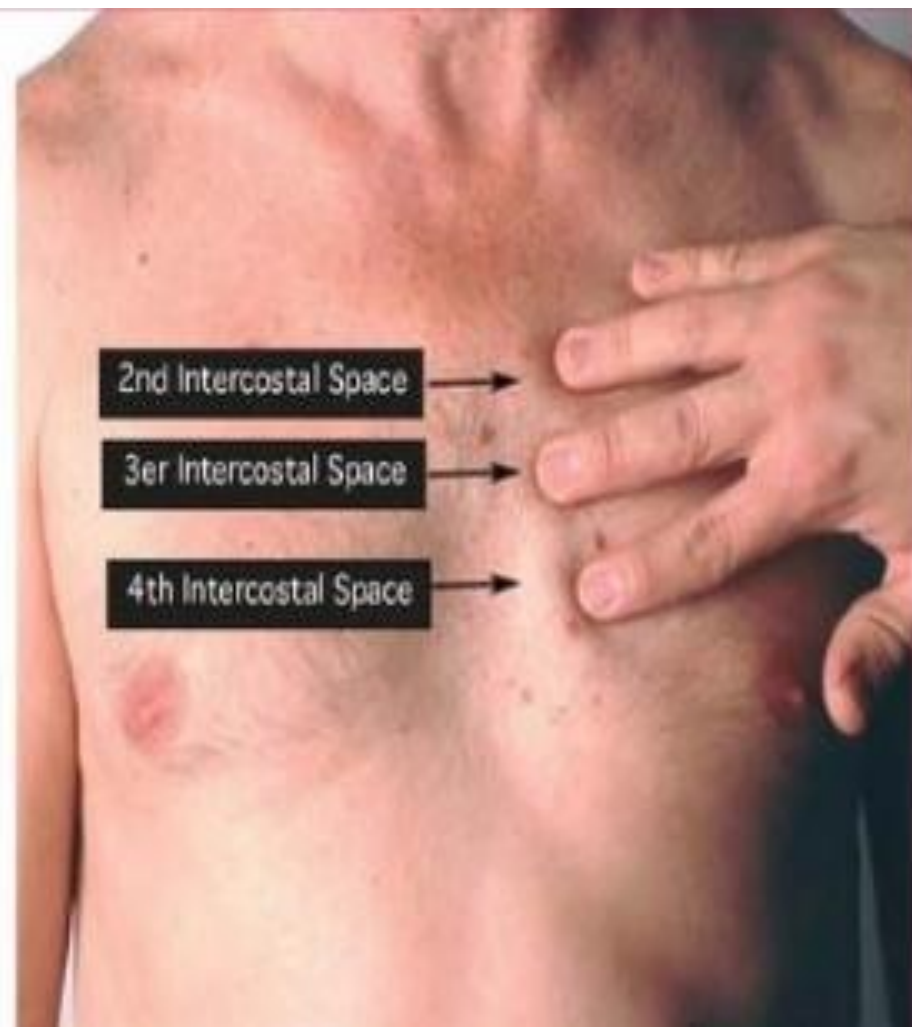


Now with the thumb or index finger of left hand please first identify the sternal angel .

if u rub your finger from manubrium sternum to downward u will find ridge or elevated area –it is the sternal angle

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Go left laterally & you will find 2<sup>nd</sup> rib  
and below its 2<sup>nd</sup> inter costal space  
and count the space to up to apex  
beat where your finger is still placed

3<sup>RD</sup>

2<sup>ND</sup>

sternal angle

2<sup>nd</sup> rib  
1<sup>st</sup> ICS

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A close-up photograph of a person's back with a hand placed on it. The fingers of the hand are numbered 2, 3, 4, and 5 from the thumb to the pinky. A black letter 'A' is placed next to the index finger. The hand is positioned over the lower back area. The person's skin is light brown. The background is a dark, textured surface, possibly a chair or table.

**A 5**  
**4**  
**3**  
**2**

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apex beat

5<sup>th</sup>

apex beat is in left 5th ICS

4<sup>th</sup> ICS

3<sup>rd</sup> ICS

2<sup>nd</sup> ICS

sternal angle

2<sup>nd</sup> rib

1<sup>st</sup> inter costal space (ICS)

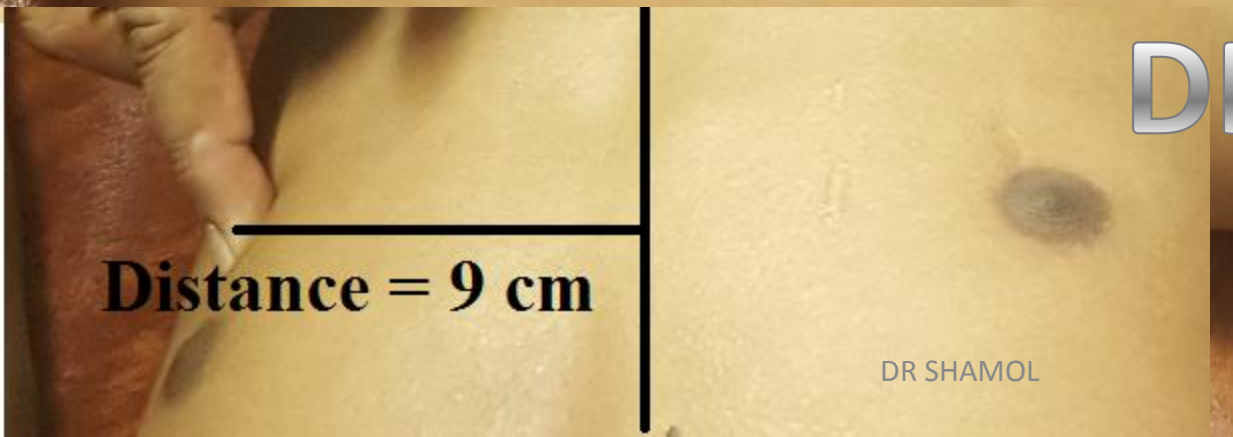
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Now take the measuring tap and measure the distance from mid line to apex beat in cm



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Remember we will see

1. site—space,
2. distance---away from mid line and
3. character

**DISTANCE**

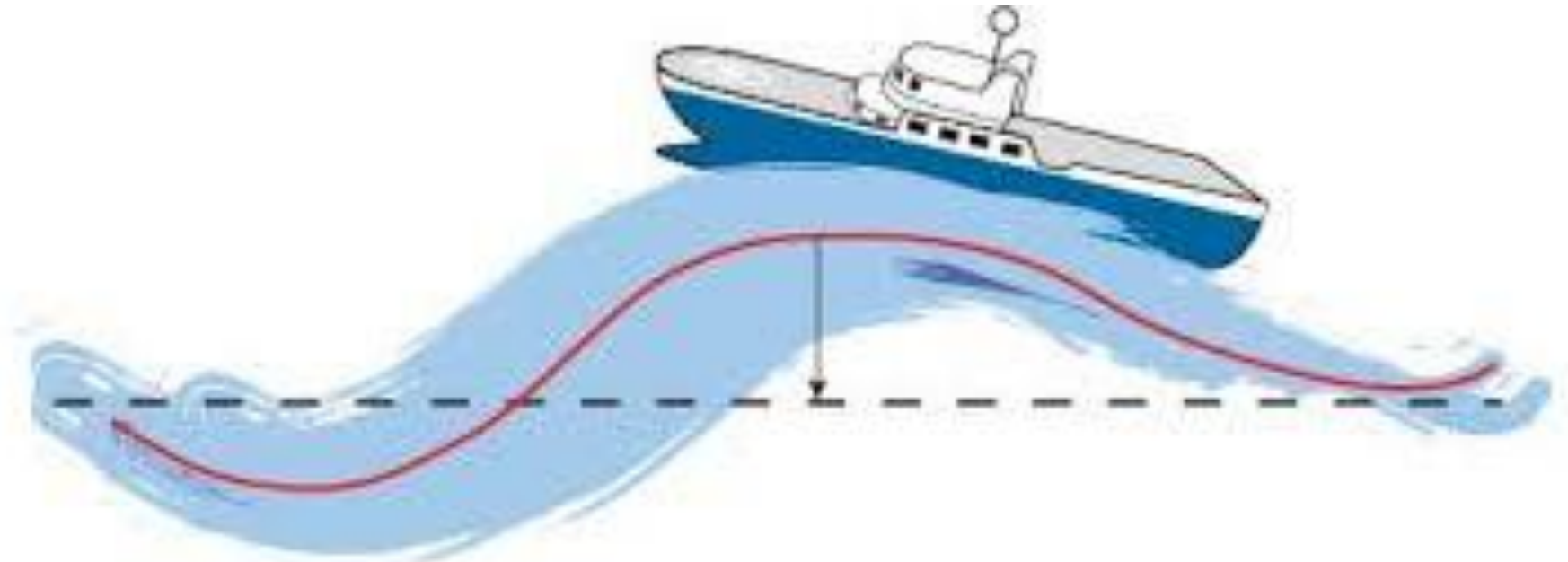
**characters**

**SITE**

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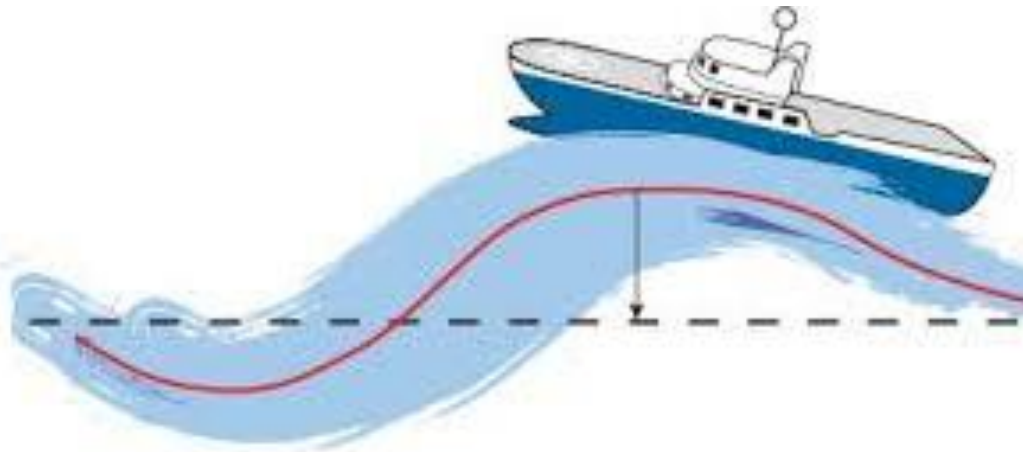
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লিমিটেড



**Left para sternal heave**





## Left para sternal heave

**place your palmar surface of right  
hand or ulnar border of right hand  
over left parasternal region**

**if left para sternal heave present  
then u will find**  
this is a sustained, thrusting pulsation  
usually felt at the left sternal edge

**What is the cause ?**  
indicating right ventricular enlargement



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**Place your palmer surface right of hand on left parasternal area & Ask the patient to hold his breath in expiration**



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**Place ulnar border of right hand over  
left parasternal area**

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ALEXANDRE AJA PRÉSENTE



Look for  
PALPABLE

P2

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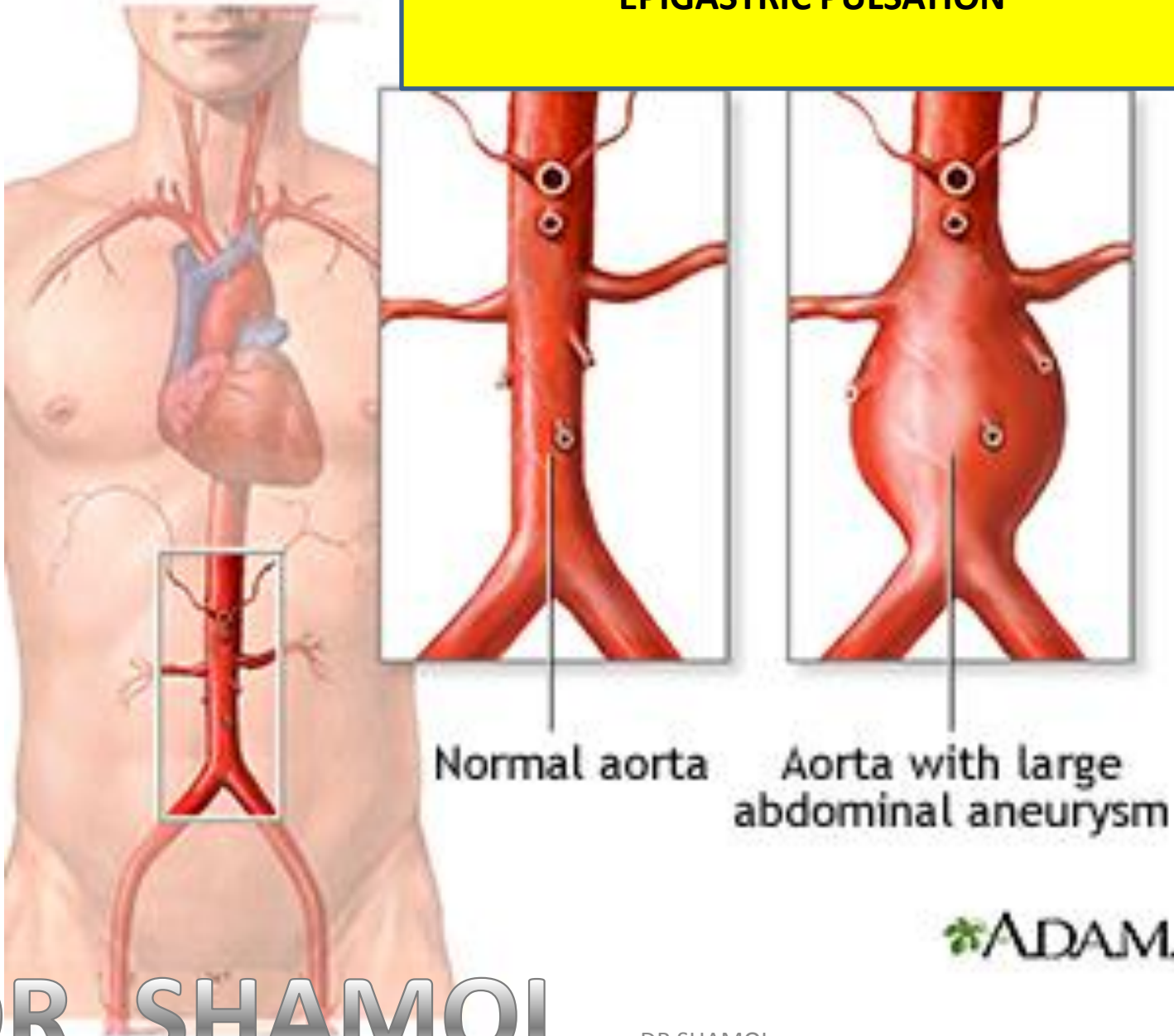


NOT SEEN NOW A  
DAYS

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Look for  
PALPABLE **A2**

## EPIGASTRIC PULSATION



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# Thrill



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## Thrill

**Palpable murmur is called thrill**

How its feel?

It feels rather like placing your hand on a purring cat

Which part of hand is used to see thrill?

Ball of finger.

Patient will be in lying position  
we examine the thrill in 4 area

1. Mitral
2. Tricuspid or left lower sternum or sternal edge
3. Aortic area
4. Pulmonary area

1. we use ball of fingers of right hand to palpate it
2. During palpation of thrill keep Ur left thumb on carotid pulse.
3. Now start from mitral area if found then roll the patient in left lateral position
4. Now place your hand in lateral to left lower sterum or tricuspid area
5. Now see thrill with ball of finger of right hand over aortic area & pulmonary area

If u got thrill in mitral area , u have to see it in left lateral position in breath hold expiration .

If u got thrill in in aortic and tricuspid area , Sit down the patient and see it in leaning forward & birth hold expiration





See thrill with  
ball of finger  
We prefer this



or

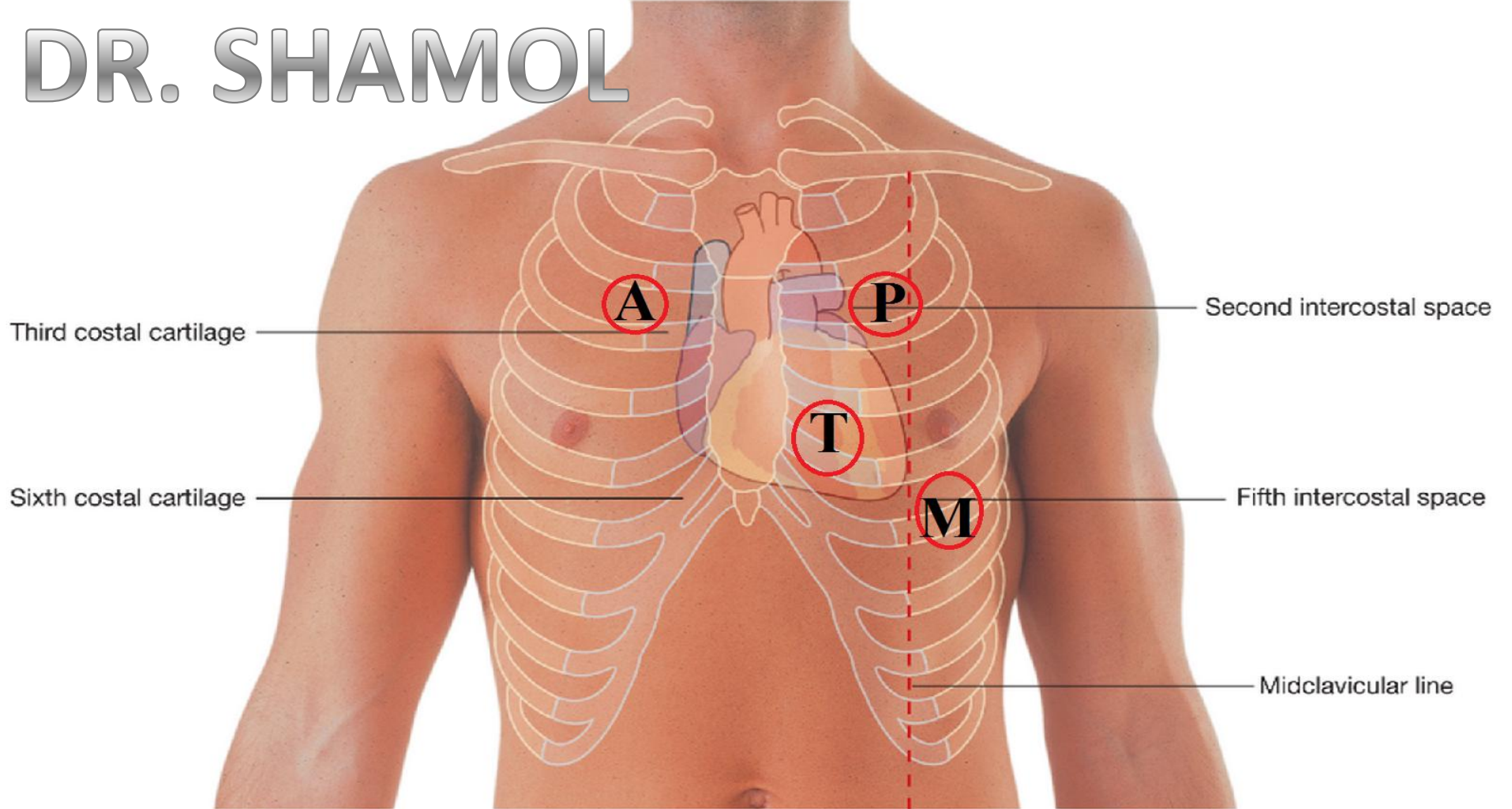


See thrill with  
heel of hand.  
Not liked by  
some examiner



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area	Location
Mitral	Left 5 <sup>th</sup> intercostal space in the mid-clavicular line (the apex).
Tricuspid	: 3 <sup>rd</sup> and 4 <sup>th</sup> intercostal space at the left sternal edge/ para sternal area
Pulmonary	: 2 <sup>nd</sup> intercostal space at the left sternal edge.
Aortic	: 2 <sup>nd</sup> intercostal space at the right sternal edge



2nd right  
interspace –  
**aortic area**

2nd left  
interspace –  
**pulmonic area**

Lower left  
sternal border –  
**tricuspid area**

Apex –  
**mitral area**

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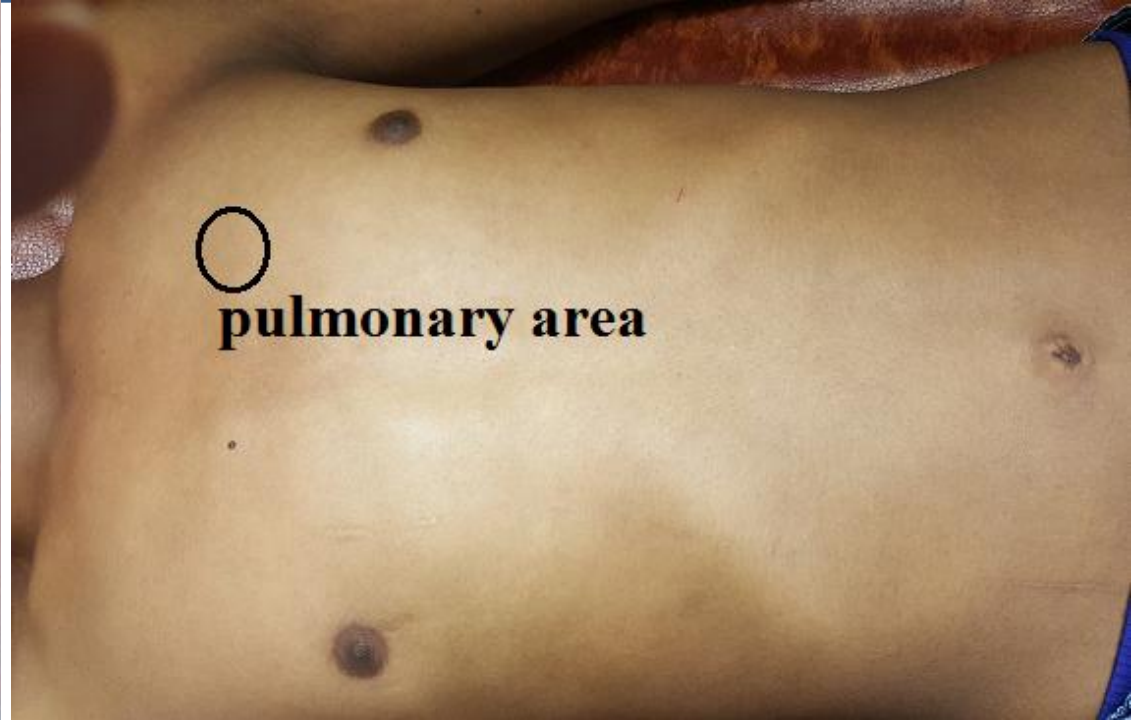
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If you get a murmur then do the following  
Keep your thumb in carotid pulse to differentiate it Systolic  
or diastolic thrill

if thrill in aortic or pulmonary and left lower sternal area  
then you have to ask the patient to sit and leaning forward  
with breath hold expiration

If mitral area then you have to roll the patient left lateral  
position and breath hold expiration

If you not get murmur then go to auscultation next





**If u got thrill in in aortic and tricuspid area ,  
Sit down the patient and see it in leaning forward & birth hold expiration**

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If u got thrill in mitral area , u have to see it in left lateral position in breath hold expiration .  
Keep left thumb on Carotid Pulse .

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# AUSCULTATION



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# Auscultation of precordium

(What will u see in auscultation of cardiovascular system)

Heart sound	Added sound
<p>Normal heart sound</p> <ul style="list-style-type: none"><li>● 1<sup>st</sup> heart sound</li><li>● 2<sup>nd</sup> heart sound</li></ul> <p>Abnormal heart sound</p> <ul style="list-style-type: none"><li>● 3<sup>rd</sup> heart sound</li><li>● 4<sup>th</sup> heart sound</li></ul>	<ul style="list-style-type: none"><li>● . Murmur</li><li>● Opening snap.</li><li>● Ejection clicks</li><li>● Pericardial rub</li></ul>

If you are asked to do only auscultation then you have to locate apex beat first to identify the mitral area. but here no need to count space and measure distance from the mid line

Name of the area	Location
Mitral	Left 5 <sup>th</sup> intercostal space in the mid-clavicular line (the apex).
Tricuspid	: 3 <sup>rd</sup> and 4 <sup>th</sup> intercostal space at the left sternal edge/ para sternal area
Pulmonary	: 2 <sup>nd</sup> intercostal space at the left sternal edge.
Aortic	: 2 <sup>nd</sup> intercostal space at the right sternal edge

2nd right  
interspace –  
**aortic area**

2nd left  
interspace –  
**pulmonic area**

Lower left  
sternal border –  
**tricuspid area**

Apex –  
**mitral area**

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1. during all stage of auscultation left thumb should always remain in right carotid artery

1. diaphragm is used in all stage of auscultation except the two condition where bell is used

- one to see the MDM of MS
- to see carotid bruit

1. All heart sound listen in lying position. No need to sit the patient if no added sound / murmur

1. Murmur is listen in two position

- In lying position ---(1)Mitral area \_\_\_MS & MR and (2) Tricuspid area\_\_\_TR and VSD
- In sitting position ----(1) Aortic are –AS , (2) Tricuspid area –AR , (3) pulmonary area –PS
- Murmur of MS seen in left lateral position

1. All the murmur listen in breath hold expiration expect murmur in pulmonary area (PS)which hard breath hold inspiration

1. Radiation seen in two area in two murmur

- In mitral area –in murmur of MR—radiation seen from Mitral area to Axilla
- In aortic area—in murmur of AS –Radiation seen aortic area to angle of right jaw over the carotid .

- First identify the apex beat if do not do it in palpation
- now place left thumb on carotid & should always remain in right carotid artery until end of examination
- Now place your diaphragm following direction in sequentially
  1. Mitral area (at apex )
  2. Tricuspid area (3<sup>rd</sup> / 4<sup>th</sup> left intercostals space just lateral to sternum )
  3. Aortic area (right 2<sup>nd</sup> intercostals space just lateral to sternum)
  4. Pulmonary area (left 2<sup>nd</sup> intercostals space just lateral to sternum )here ask patient to take deep breath to see splitting



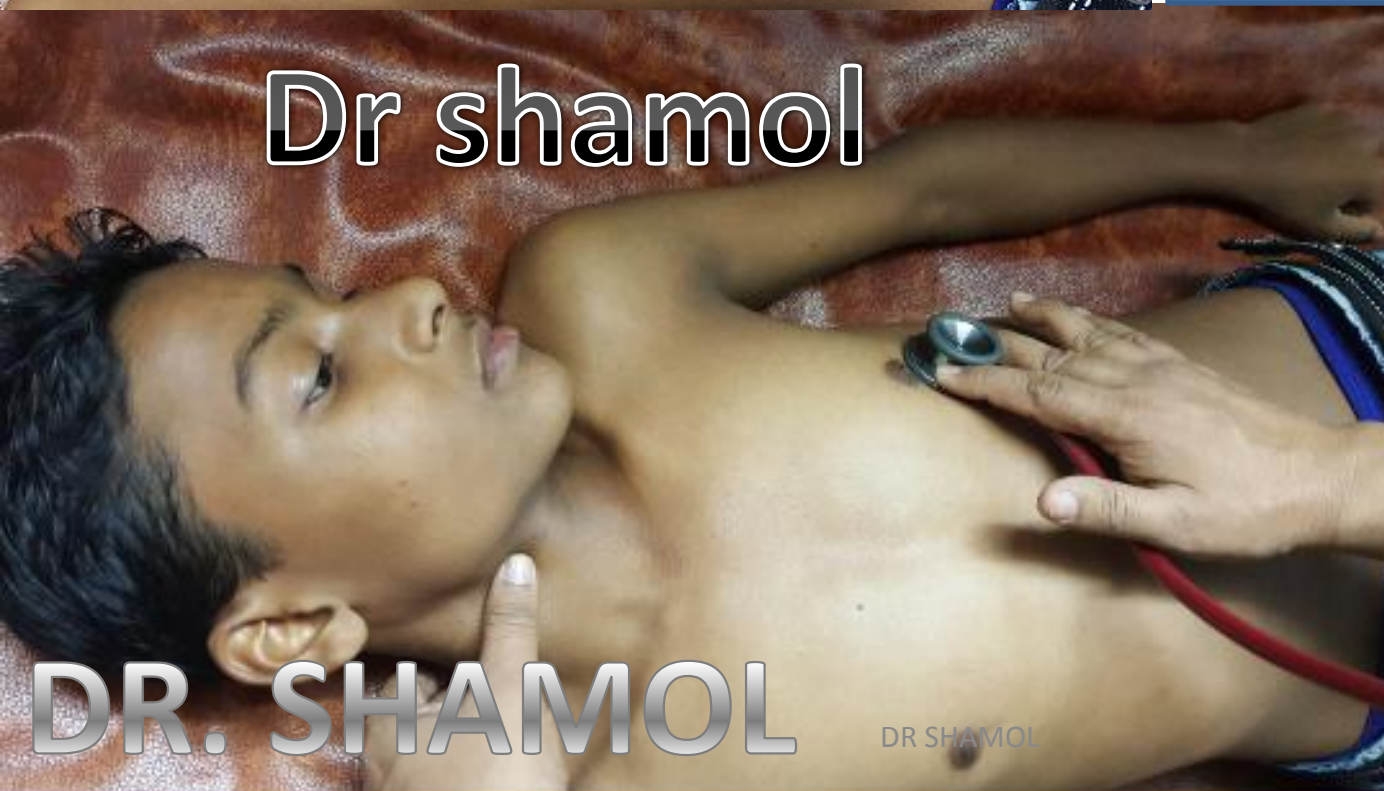


First place left thumb on right carotid artery

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- Now place your diaphragm in Mitral area (at apex )





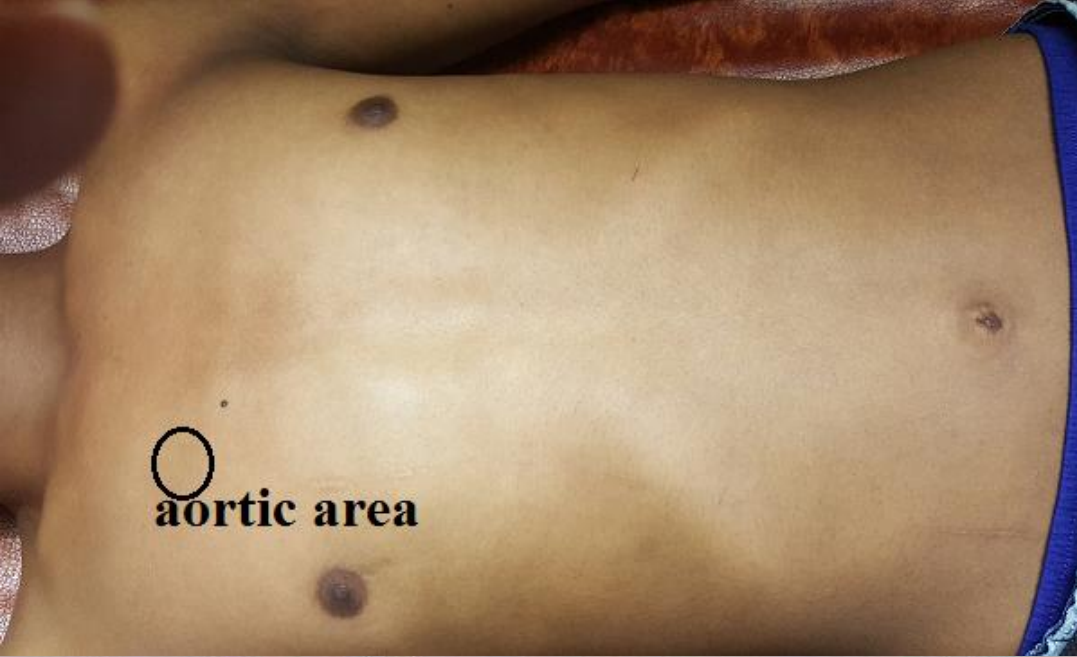


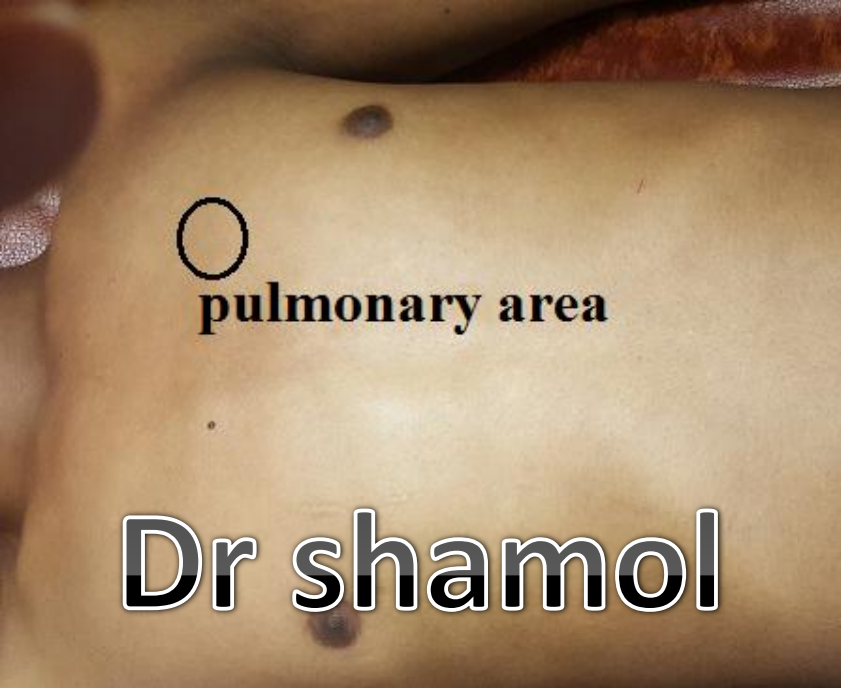
- Now place your diaphragm  
Tricuspid area (3rd / 4th left  
intercostal space just  
lateral to sternum )





- Now place your diaphragm Aortic area (right 2<sup>nd</sup> intercostals space just lateral to sternum)





○  
pulmonary area

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Now place your diaphragm  
Pulmonary area (left 2<sup>nd</sup>  
intercostals space just lateral  
to sternum ) here ask patient  
to take deep breath to see  
splitting







Auscultation position  
for aortic valve



Auscultation position  
for pulmonary valve

Aortic valve

Pulmonary valve

Tricuspid valve

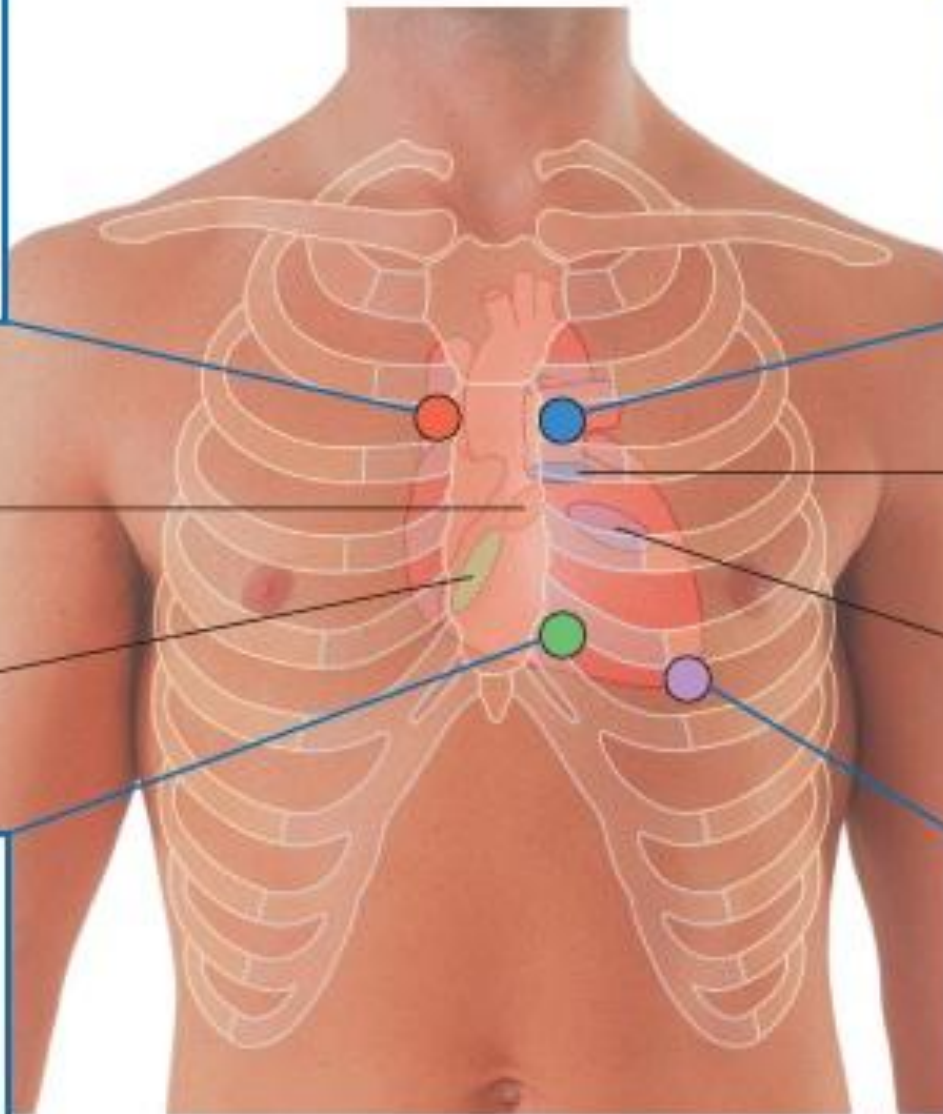
Mitral valve



Auscultation position  
for tricuspid valve

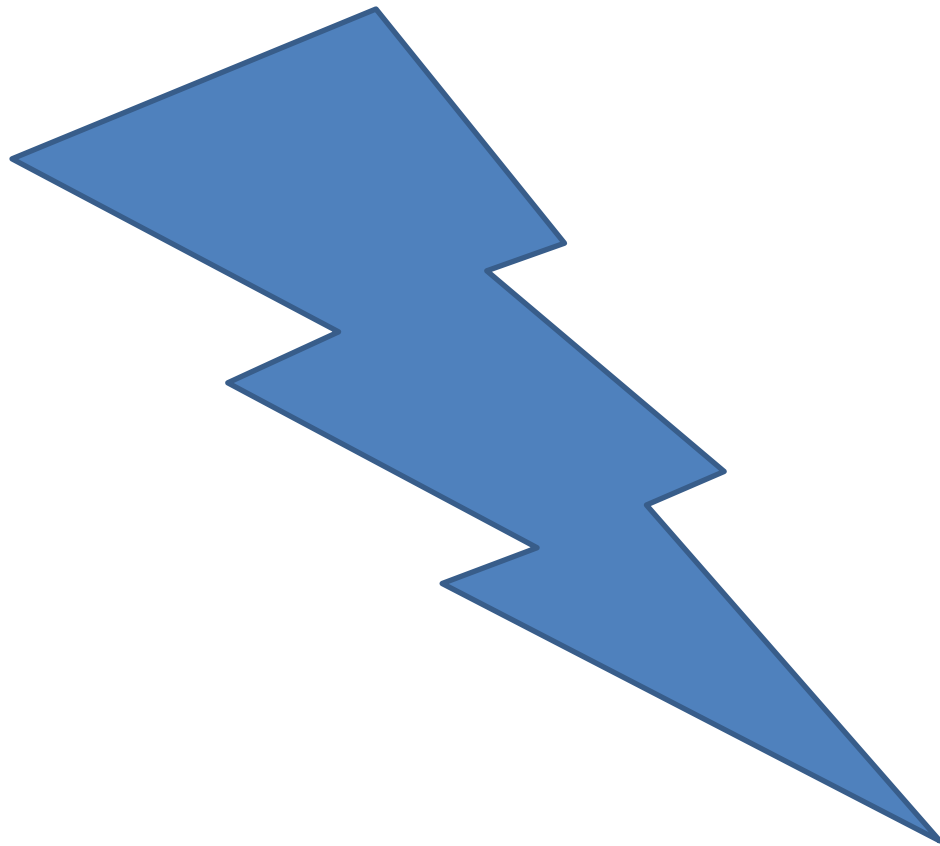


Auscultation position  
for mitral valve



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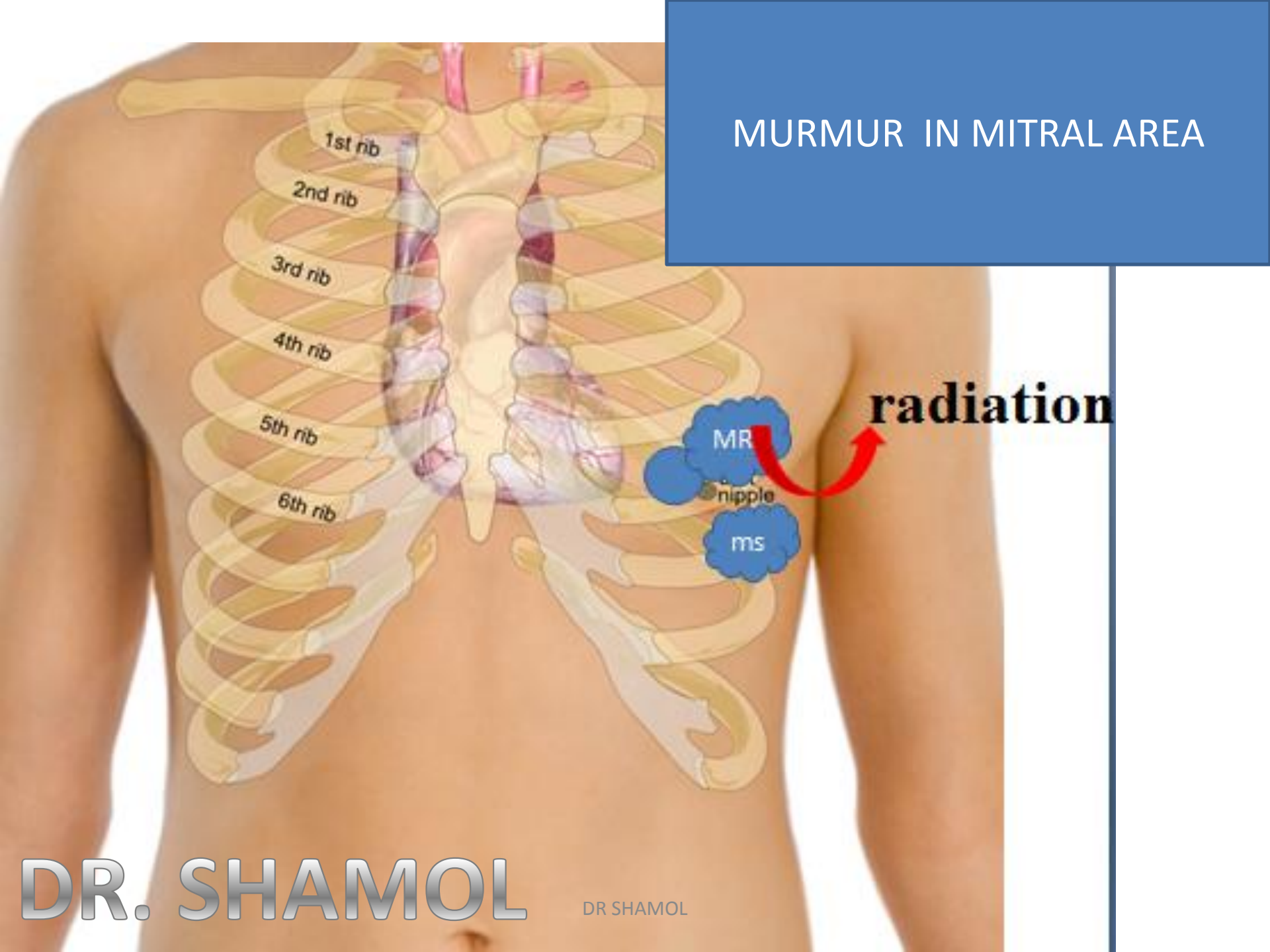




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## MURMUR IN MITRAL AREA



**radiation**

MR

nipple

ms

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# Murmur in mitral area

- First in lying position (murmur of MS & MR)
- keep left thumb should always remain in right carotid artery
- Now place your diaphragm in mitral area to see murmur if found then
  1. Roll the diaphragm and now place the bell of stethoscope on mitral area
  2. Now roll the the patient in left lateral position and ask the patient for breath hold expiration and with bell listen the murmur (MS)
  3. Now return the patient in previous position and with diaphragm see any radiation of murmur from mitral area up to axilla (MR)



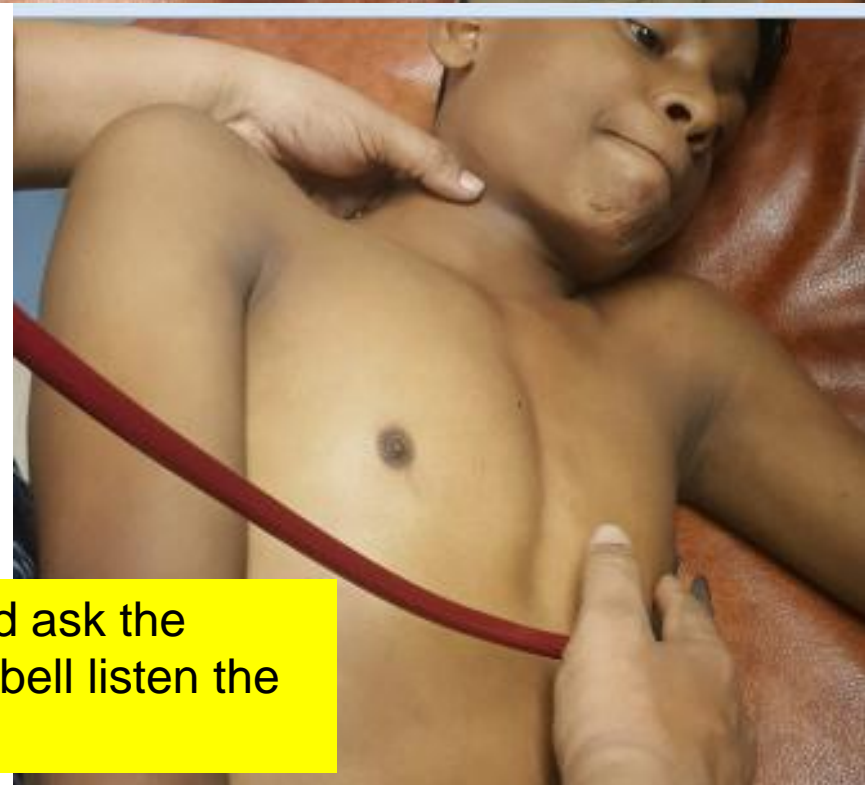
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Now turn the stethoscope the  
make the bell active

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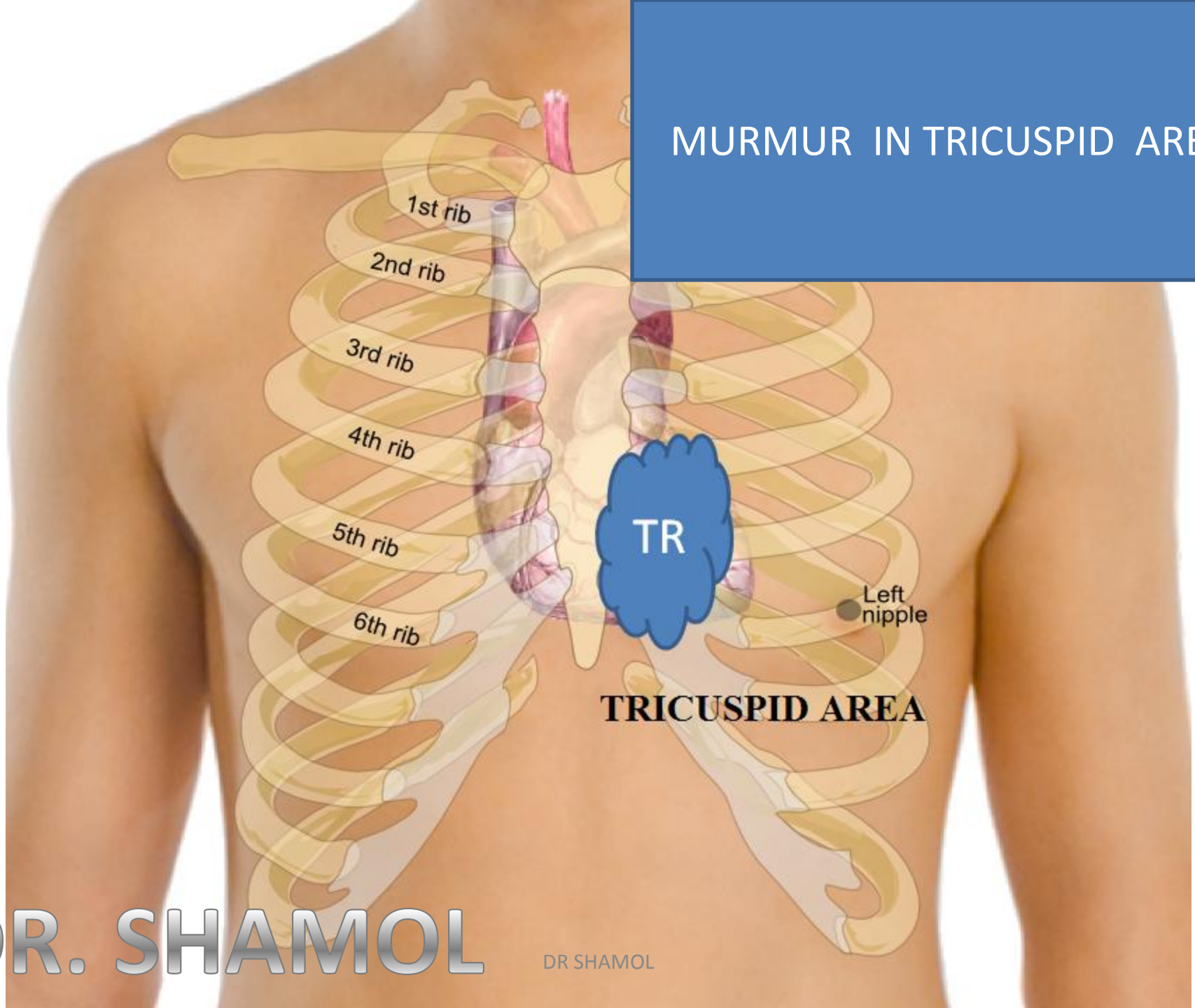




roll the the patient in left lateral position and ask the patient for breath hold expiration and with bell listen the murmur (MS)

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# MURMUR IN TRICUSPID AREA



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Murmur in tricuspid area  
Now place diaphragm of the stethoscope in tricuspid area and try to hard any murmur present or not

- Here no radiation seen
- No matter of respiration
- No posture change

Here just place the stethoscope and see any murmur present or not

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Now change the position

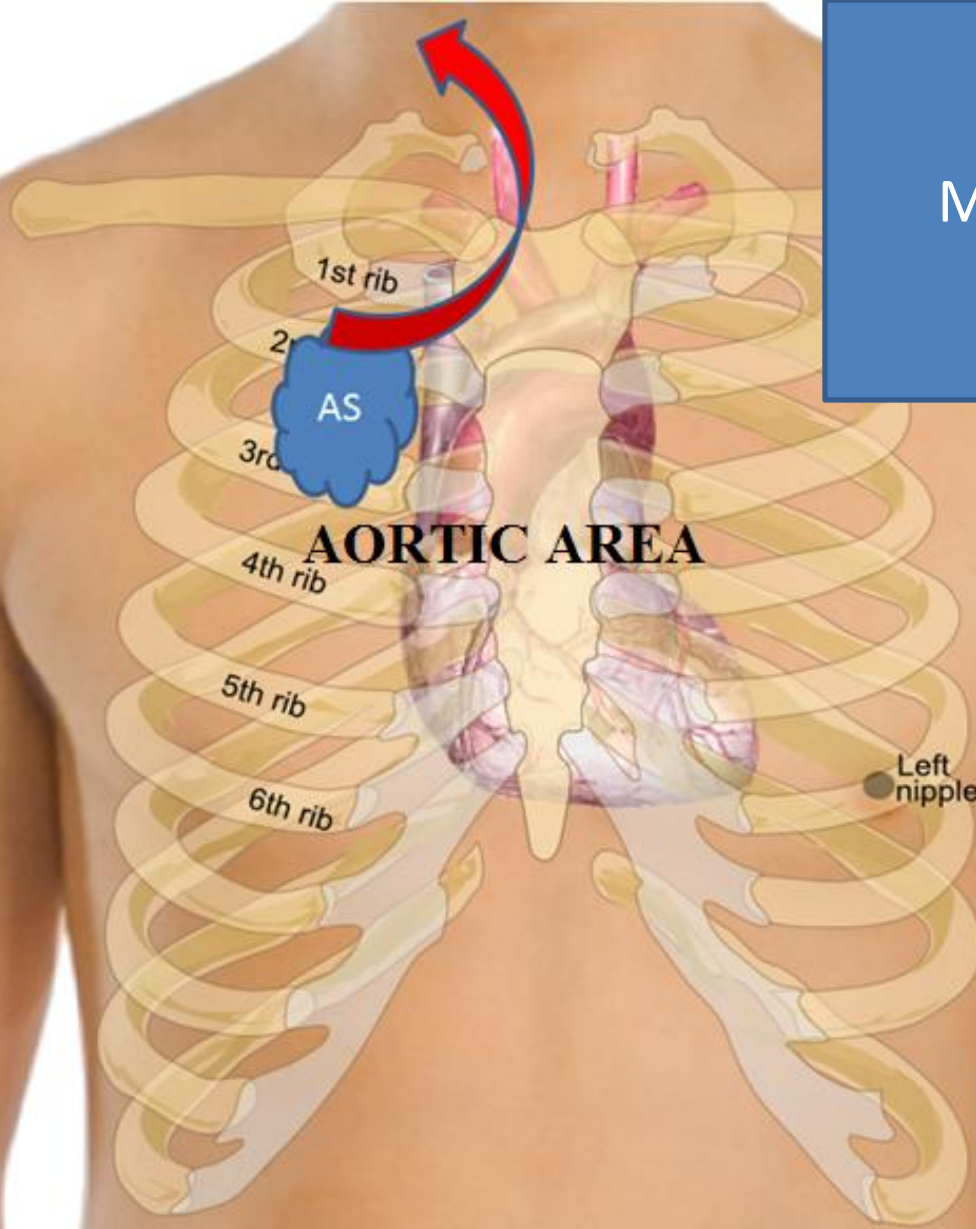


Ask the patient  
to sit from lying  
position

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## MURMUR IN AORTIC AREA



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## Aortic area

1. Now ask the patient to sit down and leaning forward
2. keep your left thumb should as usual in right carotid artery
3. Now ask the patient for breath hold expiration
4. Place diaphragm of stethoscope in aortic area
5. Now with the diaphragm see the radiation of murmur (AS) from aortic area to just below angle of right jaw over right neck
6. it better during listening radiation turn the patient s head toward the left and removed the that was over the carotid pulse



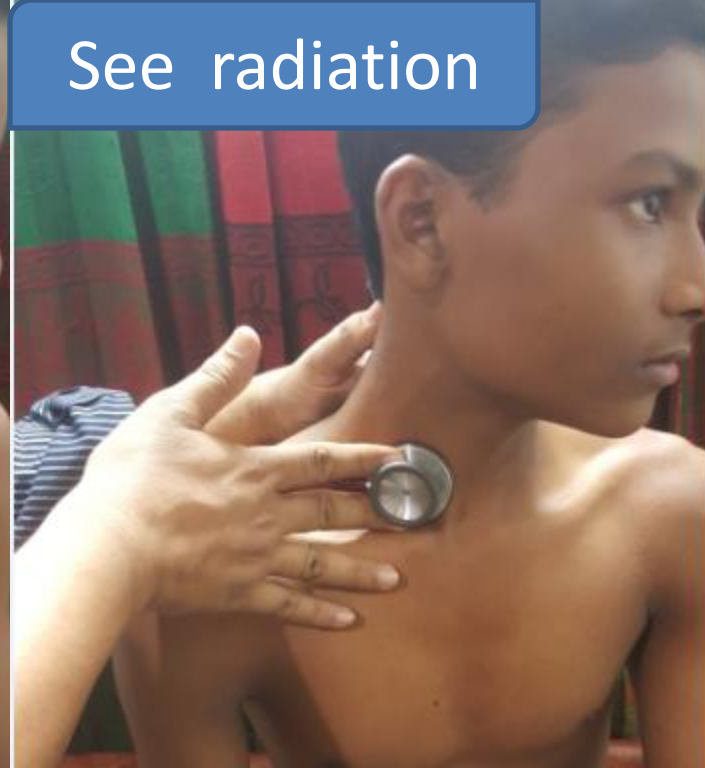


Now ask the patient to sit and leaning forward and breath hold after expiration  
Place Diaphragm On aortic area

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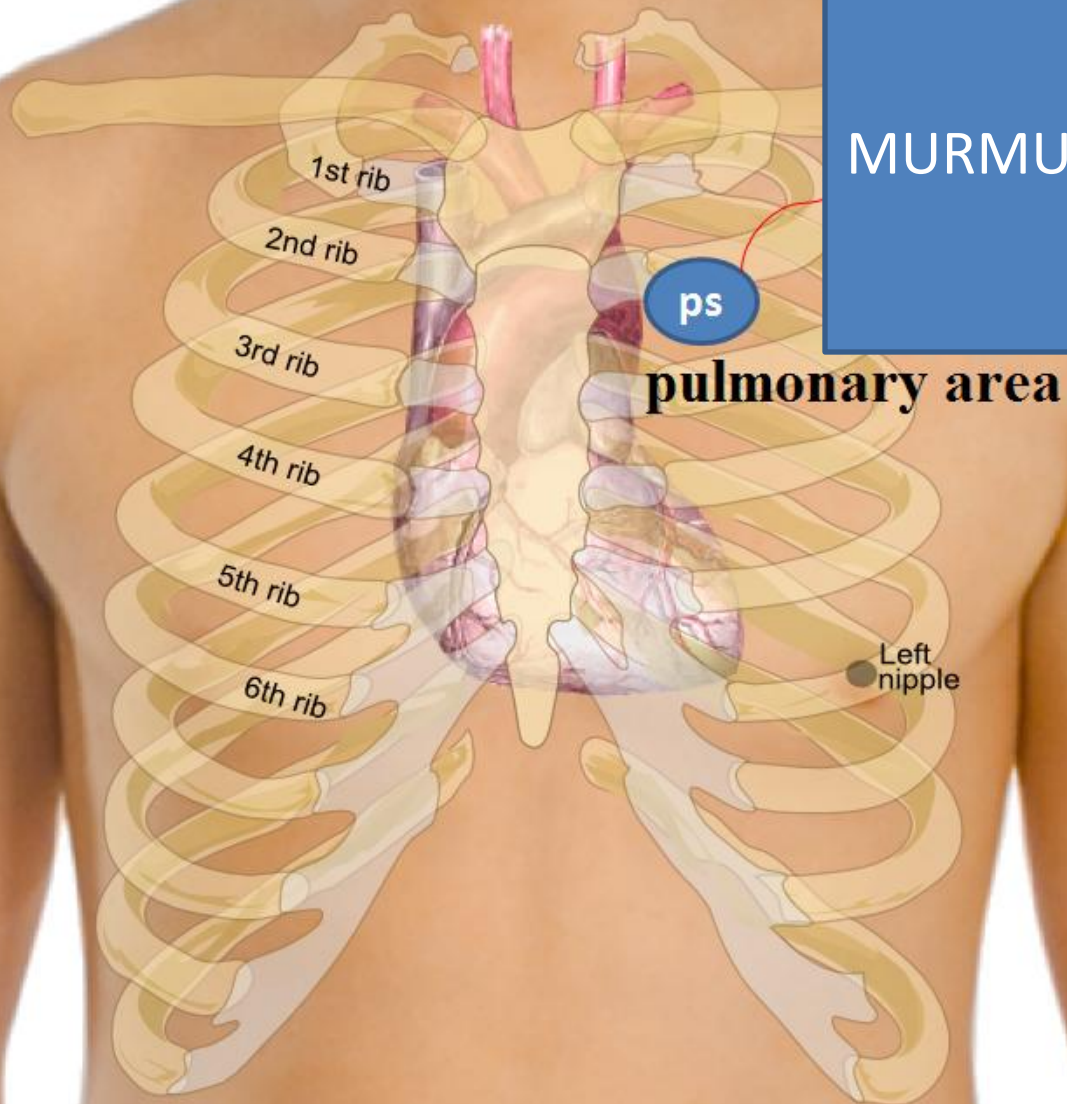
See radiation



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# MURMUR IN PULMONARY AREA



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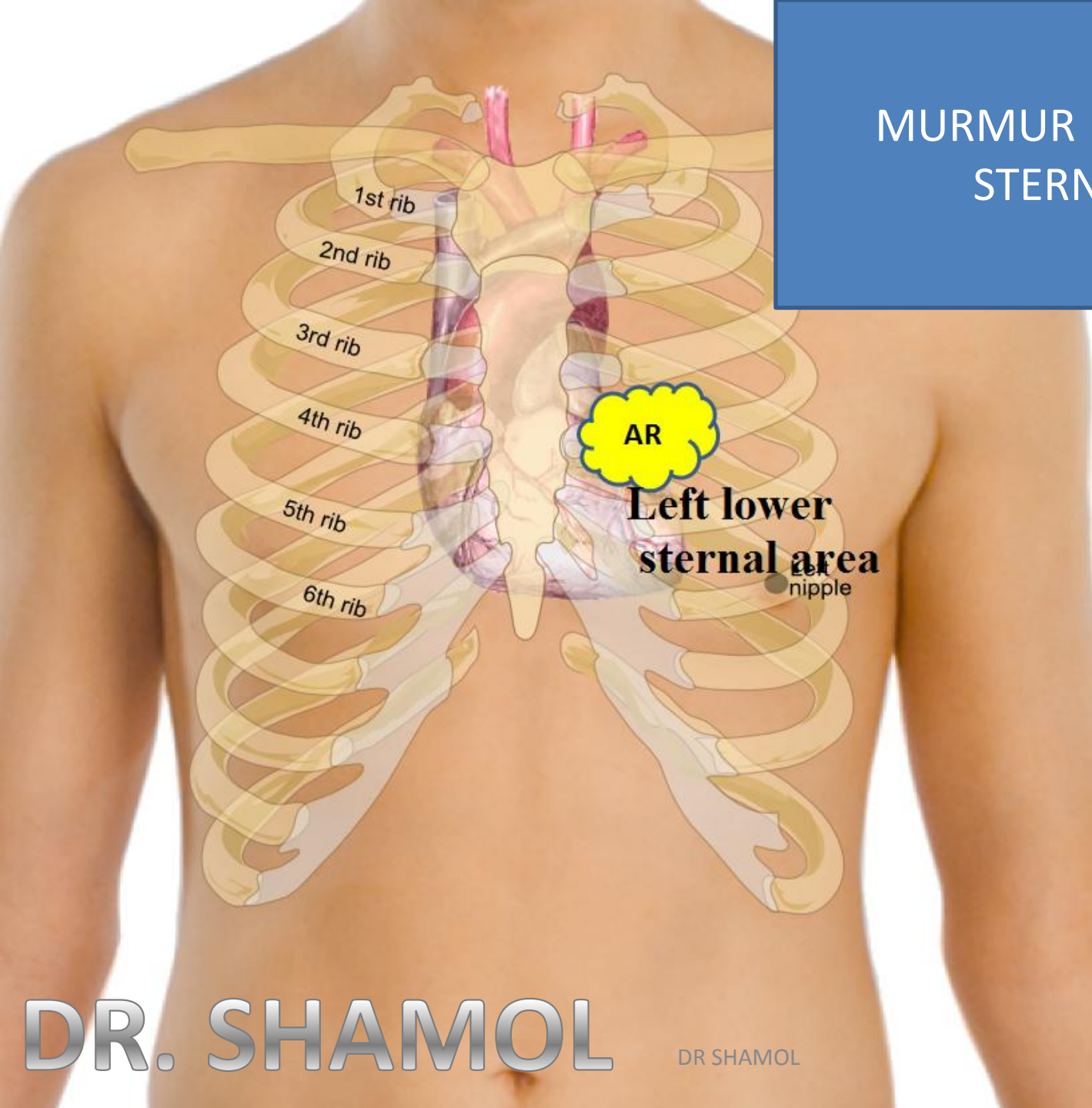
Now pulmonary area

- The patient will still sit down and leaning forward
- your left thumb also remain over the right carotid artery as usual
- Now ask the patient for breath hold inspiration
- Place diaphragm of stethoscope in pulmonary area
- listen for murmur



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# MURMUR LEFT L LOWER STERNAL AREA



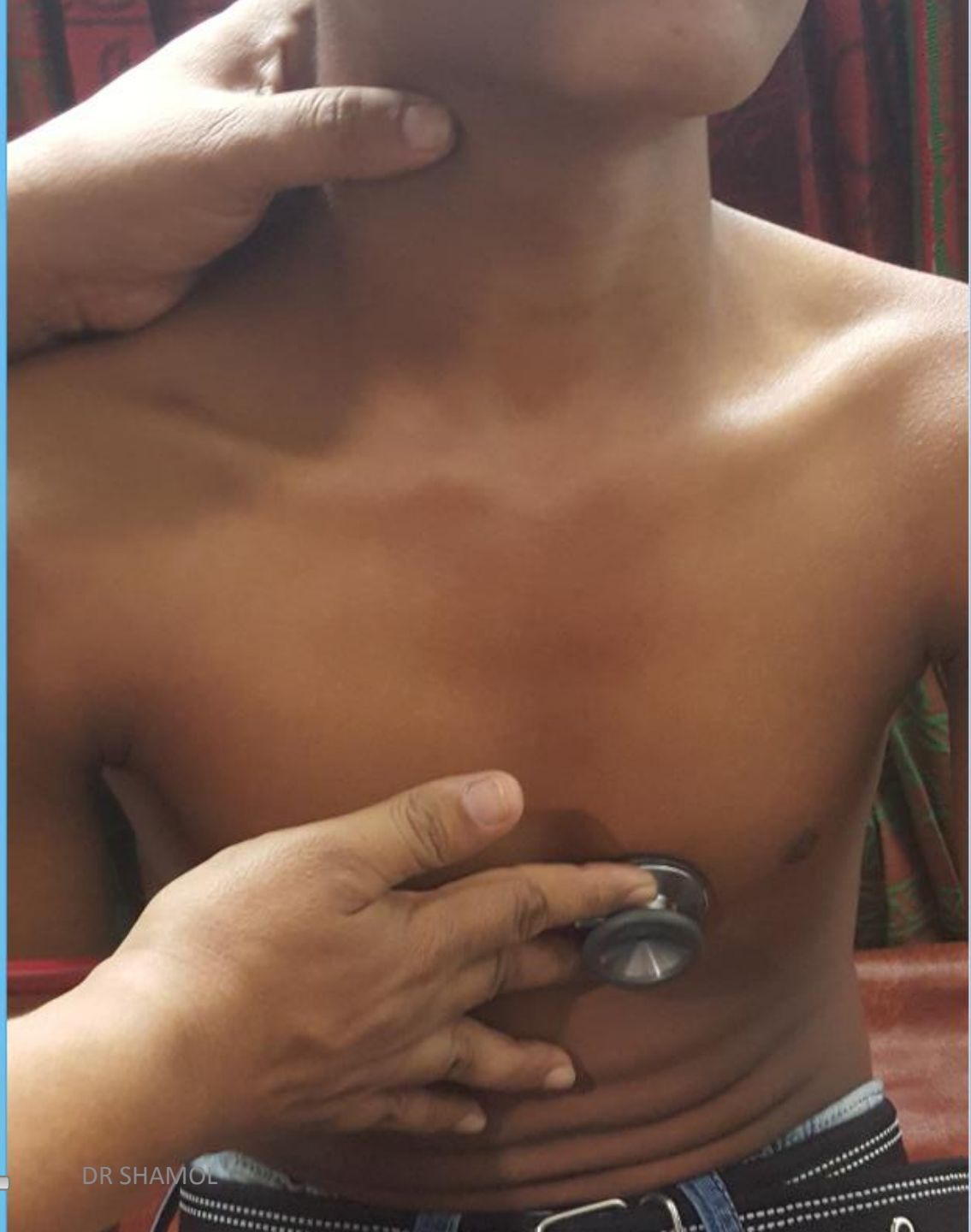
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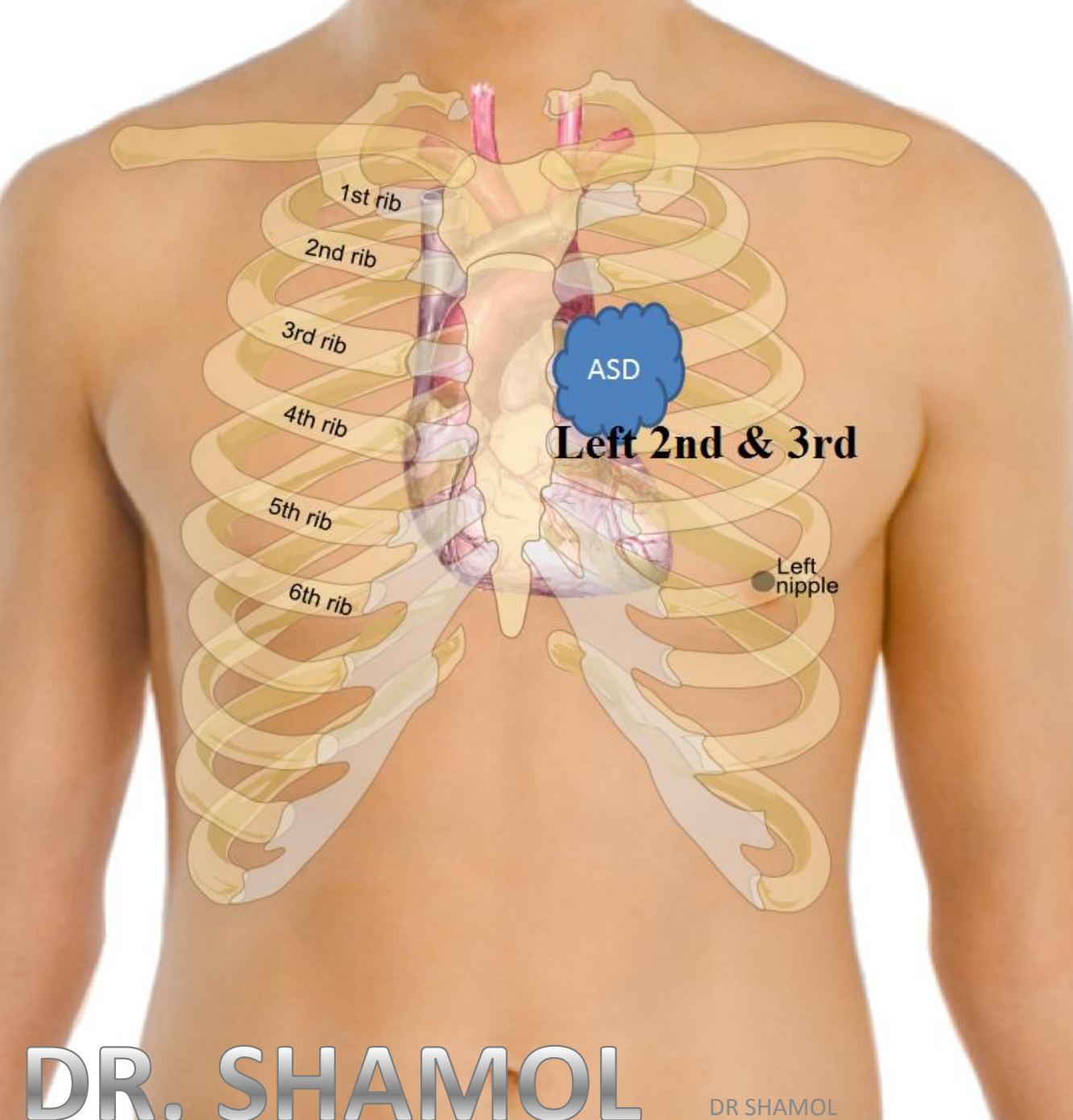
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Now left lower sternal edge

- The patient will still sit down and leaning forward
- your left thumb also remain over the right carotid artery as usual
- Now ask the patient for breath hold expiration
- Place diaphragm of stethoscope in tricuspid area
- listen for murmur



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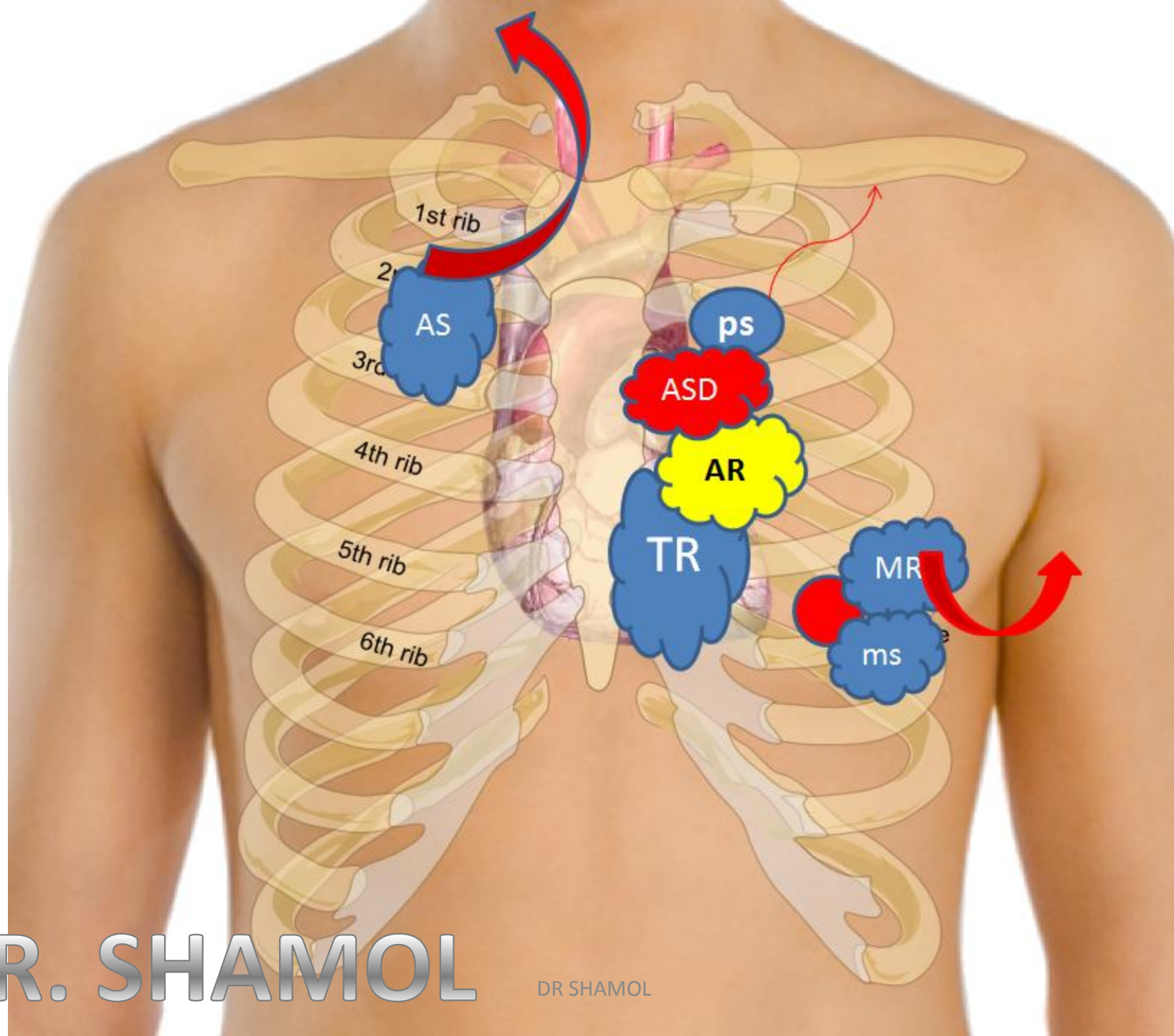


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## 6.25 Cardiac auscultation: the best sites for hearing abnormality

Site	Sound
Cardiac apex	First heart sound Third and fourth heart sounds Mid-diastolic murmur of mitral stenosis
Lower left sternal border	Early diastolic murmurs of aortic and tricuspid regurgitation
Upper left sternal border	Second heart sound Opening snap of mitral stenosis Pulmonary valve murmurs Pansystolic murmur of ventricular septal defect
Upper right sternal border	Systolic ejection (outflow) murmurs, e.g. aortic stenosis, hypertrophic cardiomyopathy
Left axilla	Radiation of the pansystolic murmur of mitral regurgitation
Below left clavicle	Continuous 'machinery' murmur of a persistent patent ductus arteriosus





MURMUR OF PDA

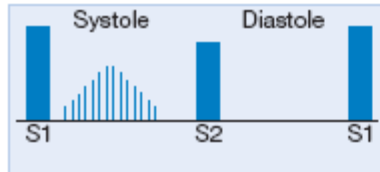
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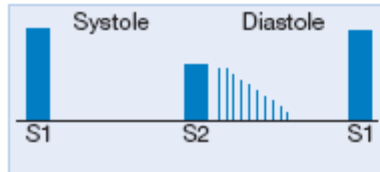
## AORTIC VALVE

③

Stenosis



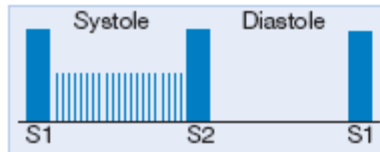
Regurgitation



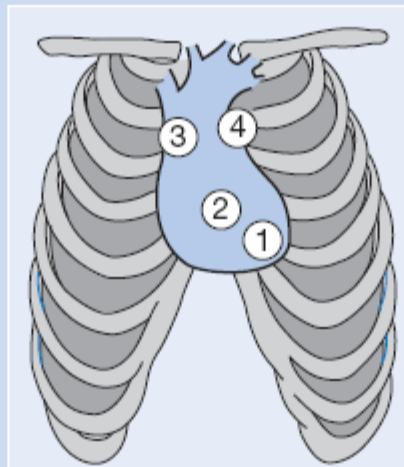
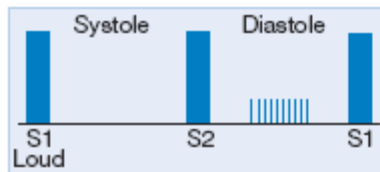
## TRICUSPID VALVE

②

Regurgitation



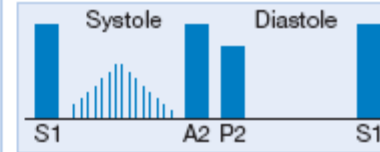
Stenosis



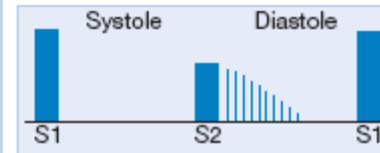
## PULMONARY VALVE

④

Stenosis



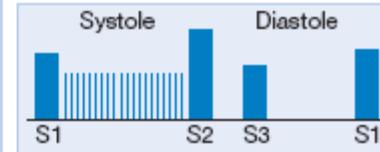
Regurgitation



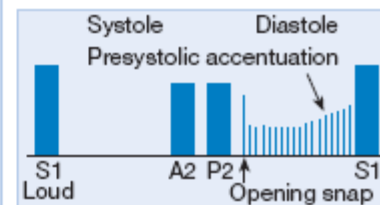
## MITRAL VALVE

①

Regurgitation



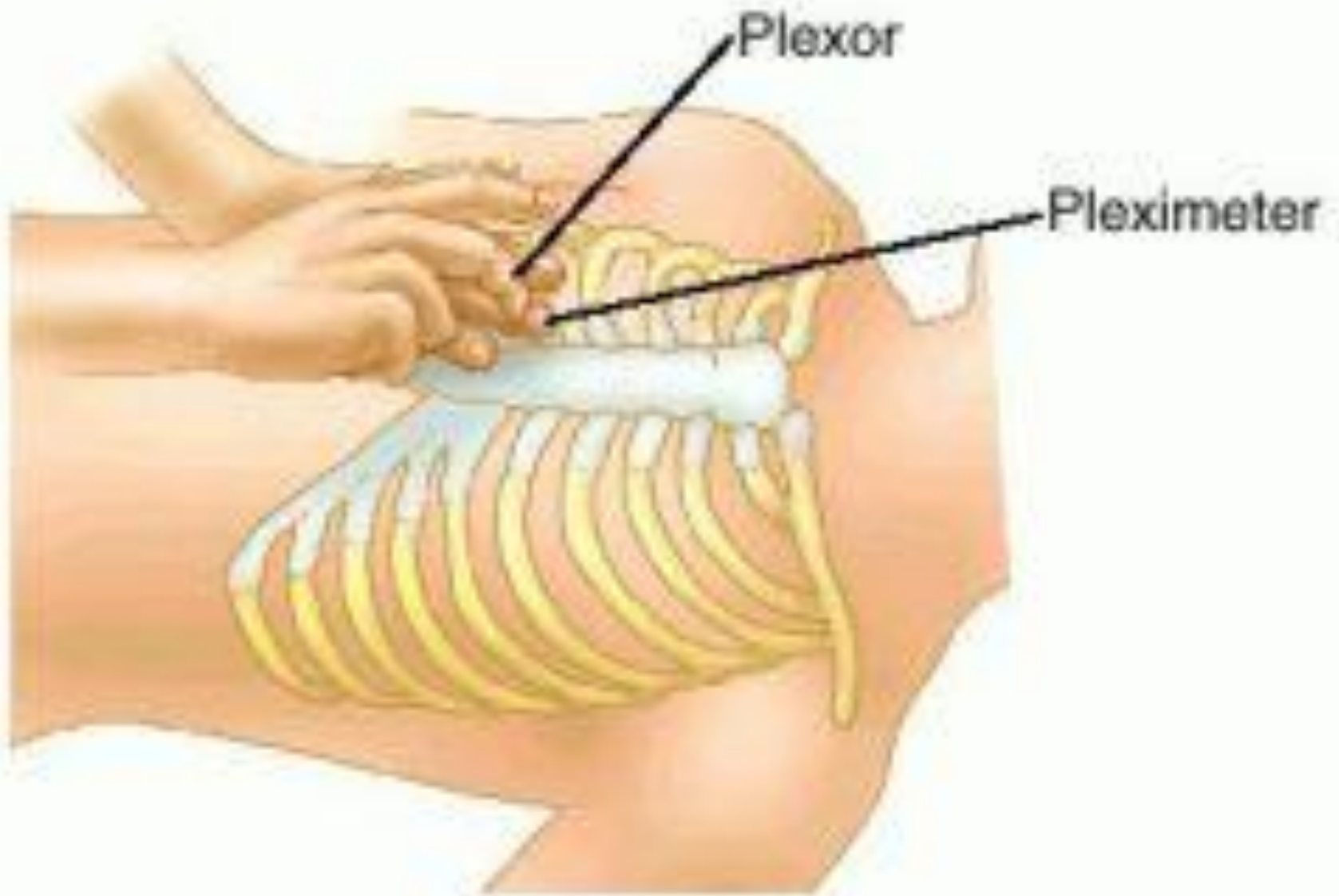
Stenosis



# Murmurs Summary

	<b>Aortic Stenosis</b>	<b>Aortic regurgitation</b>	<b>Mitral Stenosis</b>	<b>Mitral regurgitation</b>
Pulse	Slow-rising	Collapsing	Often AF	
Apex beat	Forceful, not displaced	Displaced	Tapping, not displaced	Thrusting, +/- displaced
Murmur	Ejection systolic	Early diastolic	Rumbling mid-diastolic	Pansystolic
Best heard	Aortic area	Tricuspid area	Mitral area	Mitral area
Radiation	Carotids			Axilla





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Usually it is not practice

In percussion we area of the superficial cardiac dullness

Patient should be in lying position

In this percussion we will identify the area of superficial cardiac dullness

For this u have to identify a tri angle that has three border

**Right border** of hear that is form by the right atrium ,

**base or superior** border of heart that is form by the great vessel

**Inferior border** of heart that is formed by apex or left ventricle

so u have to percussion in three direction to find out this three border

- First right border**

Percussion vertically from right nipple to midline until dullness appear . where will u get the dullness , u will immediately stop there because it will be the right border of heart

- Now base or superior border**

percussion obliquely just bellow the left shoulder toward the xephoid process until dullness appear and stop immediately when u will get the dullness and it will be the superior border or base

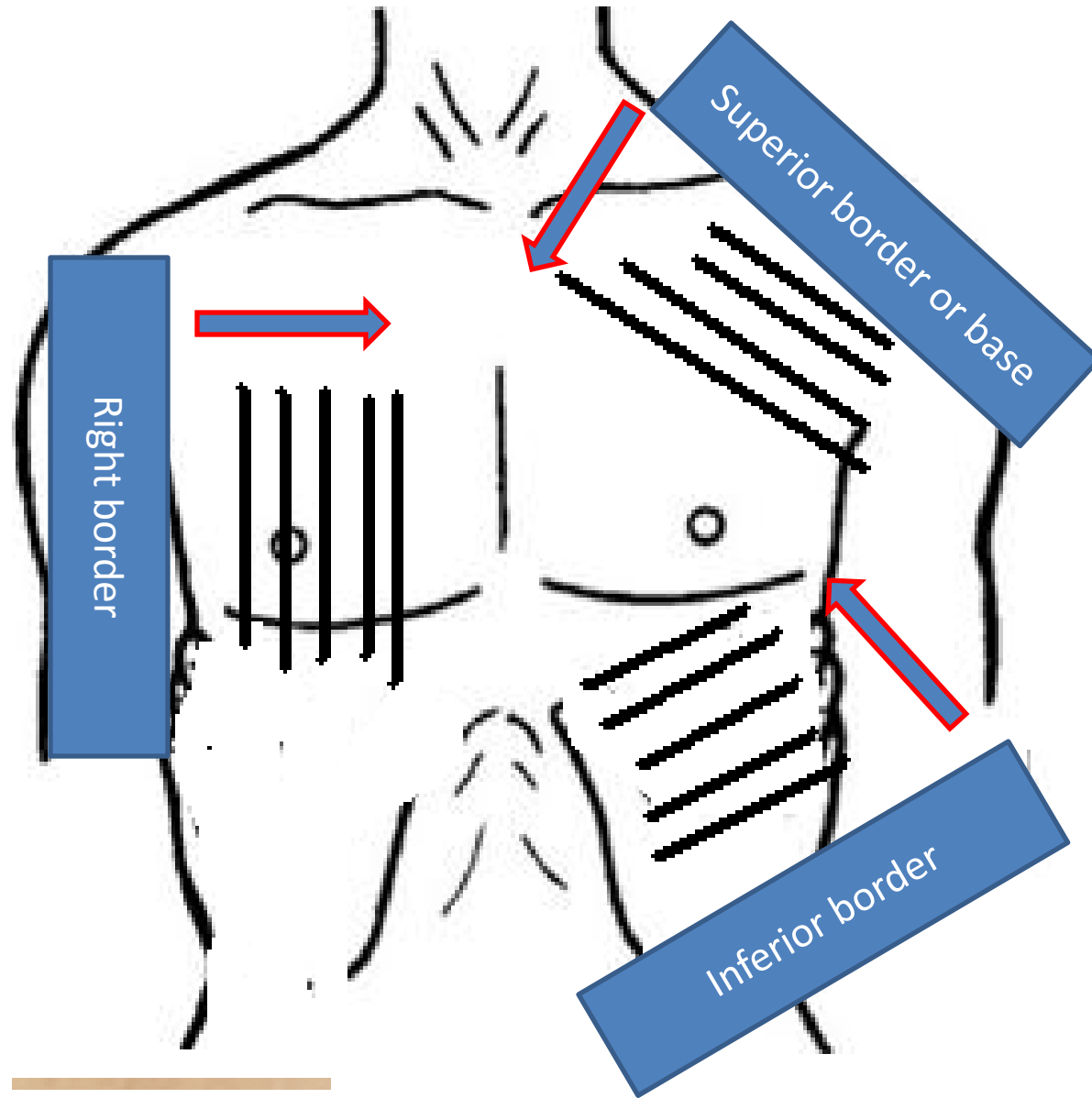
- Now see the inferior border**

percussion obliquely from left lower chest toward the left nipple or apex until dullness appear and stop immediately when u will get the dullness and it will be the inferior border of heart .

- The area among these three borders is the area of superficial cardiac dullness. after percussion u have to comment that this area of cardiac dullness is

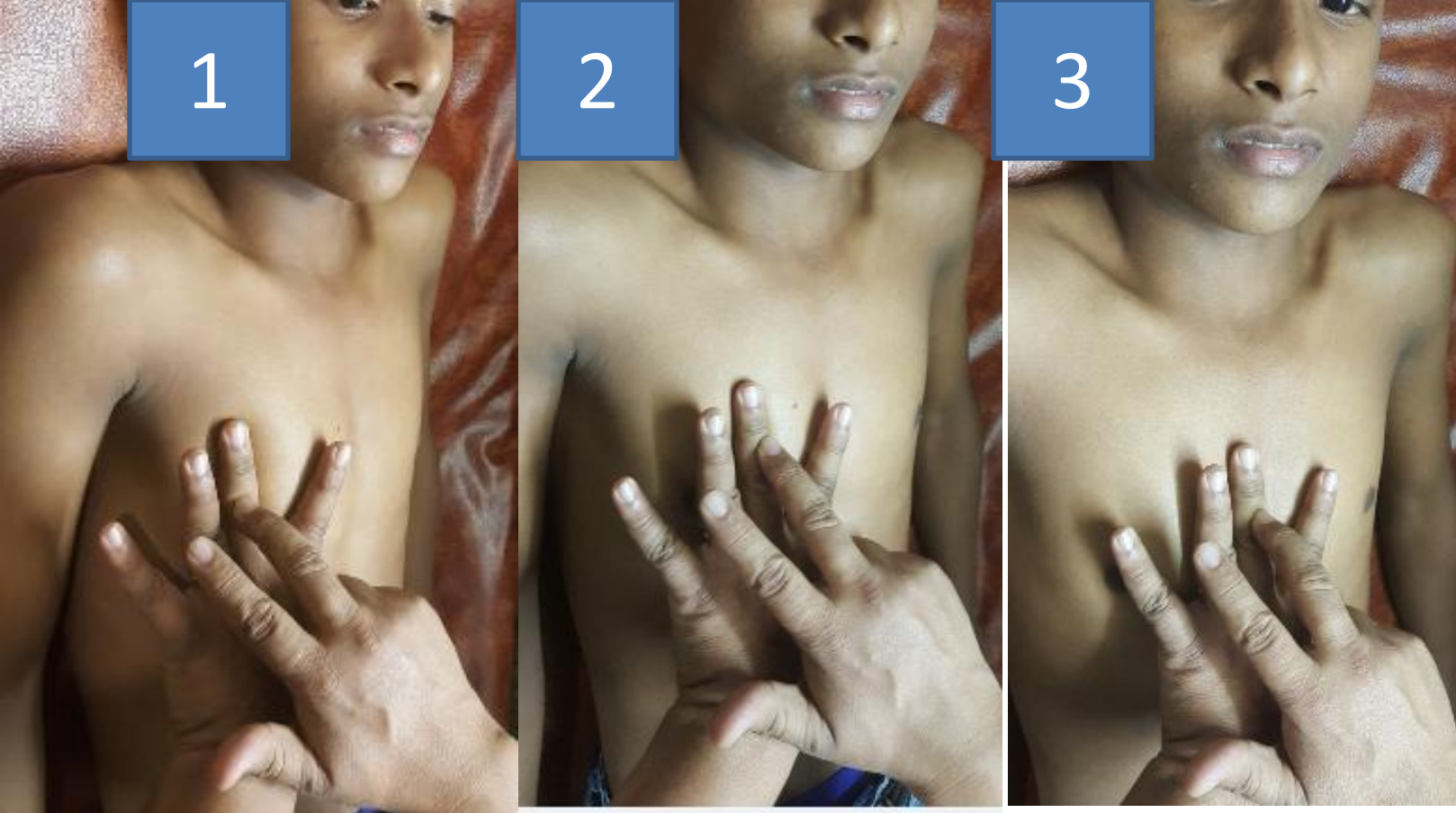
- Normal or
- Increased (in pericardial effusion )
- Decreased (emphysema )





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### •right border

Percussion vertically from right nipple to midline until dullness appear . where will u get the dullness , u will immediately stop there because it will be the right border of heart



1

A close-up photograph of a person's left upper chest. A hand is positioned with the index finger pointing towards the apex of the heart, while the other fingers are spread out. The hand is resting on the skin, which is light brown. A blue denim strap is visible at the top of the frame.



2

A close-up photograph of the same person's left upper chest. The hand is now in a percussion position, with the index finger extended and the other fingers curled. The hand is moving towards the apex of the heart. A blue denim strap is visible at the top of the frame.



3

A close-up photograph of the same person's left upper chest. The hand is now in a percussion position, with the index finger extended and the other fingers curled. The hand is moving towards the apex of the heart. A blue denim strap is visible at the top of the frame.

•**the inferior border**  
percussion obliquely from  
left lower chest toward the  
left nipple or apex until  
dullness appear and stop  
immediately when u will get  
the dullness and it will be  
the inferior border of heart

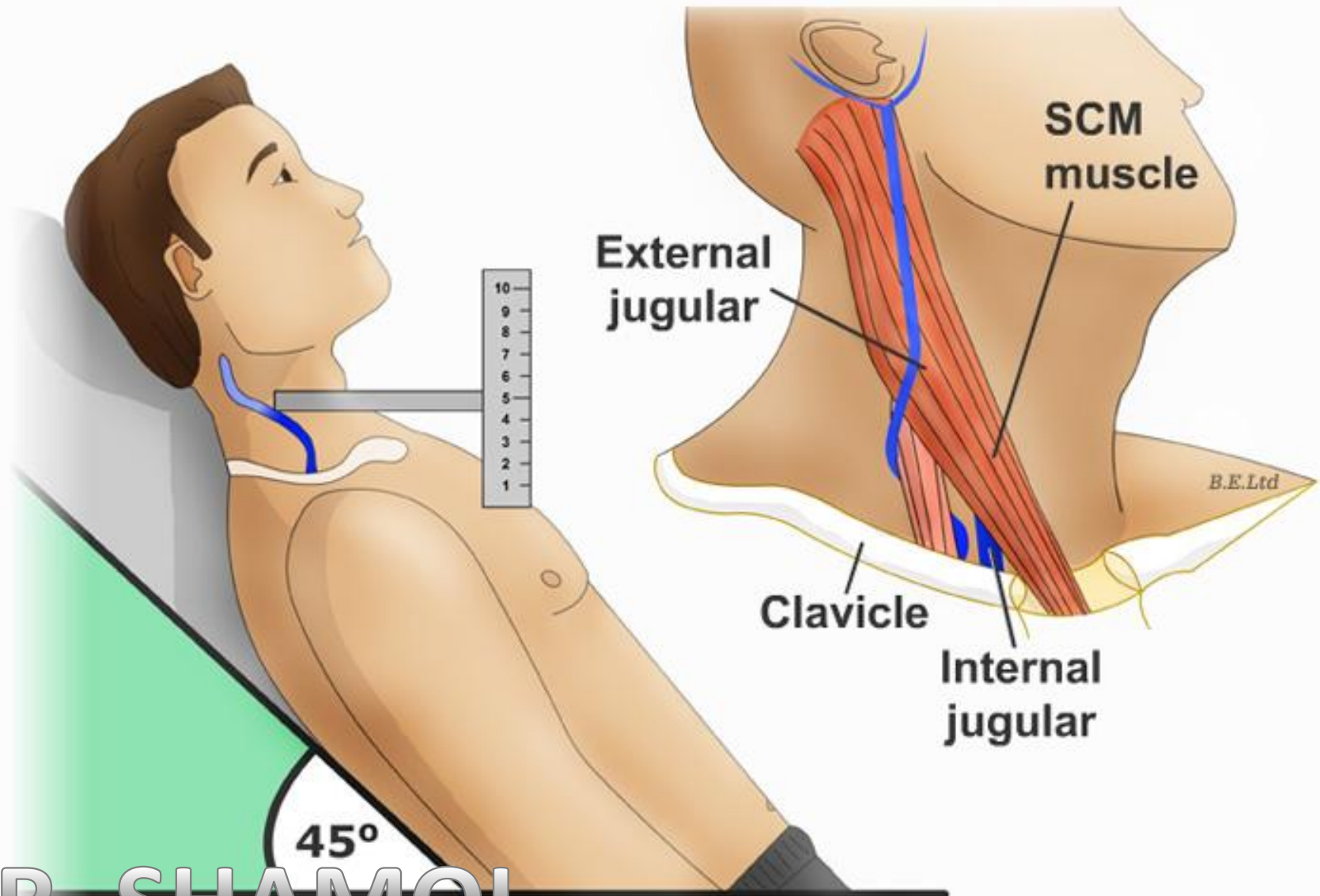




- **base or superior border**

percussion obliquely just below the left shoulder toward the xiphoid process until dullness appear and stop immediately when u will get the dullness and it will be the superior border or base

# Jugular venous pressure



- ❖ ask the examiner that I need back rest to keep the pt in 45° position
- ❖ Then the examiner will be told ok take it or ok I can not give u the back rest. pl examination with available facility
- ❖ Ensure that the neck muscles are relaxed by resting the back of the head on 1 or 2 pillow



# Patient Position in **JVP** Examination



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If pillow is not available  
Then rest the patient head and neck  
On your hand and incline  $45^{\circ}$

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Look across the neck  
from the right side of the  
patient & Identify the  
internal jugular pulsation

**External  
Jugular Vein**

**Path of IJ**

**Clavicular  
Head of SCM**

**Clavicle**

**Sternal Head of SCM**

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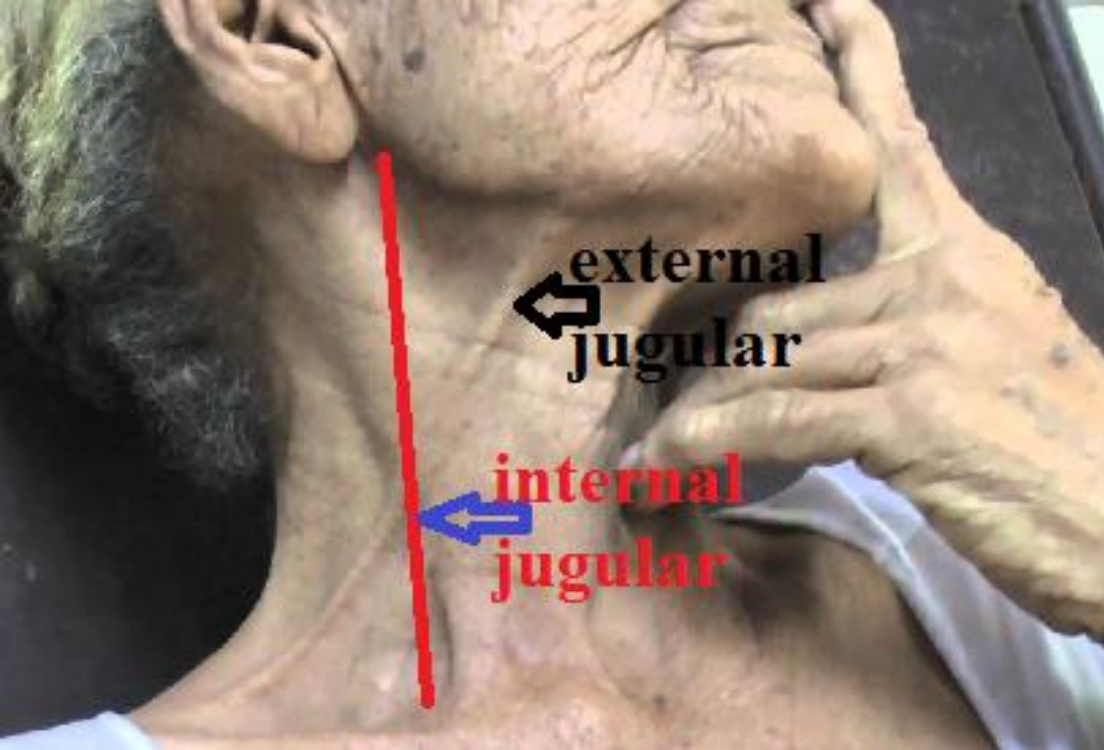
**Clavicle**

**Internal  
jugular vein**

**Sternocleidomastoid  
muscle**

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Look across the neck from  
the right side of the patient  
& Identify the internal  
jugular pulsation

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For confirmation do the abdomino-jugular reflux (keep the right hand just bellow the right costal cartilage & give upward pressure it will make JVP prominent )



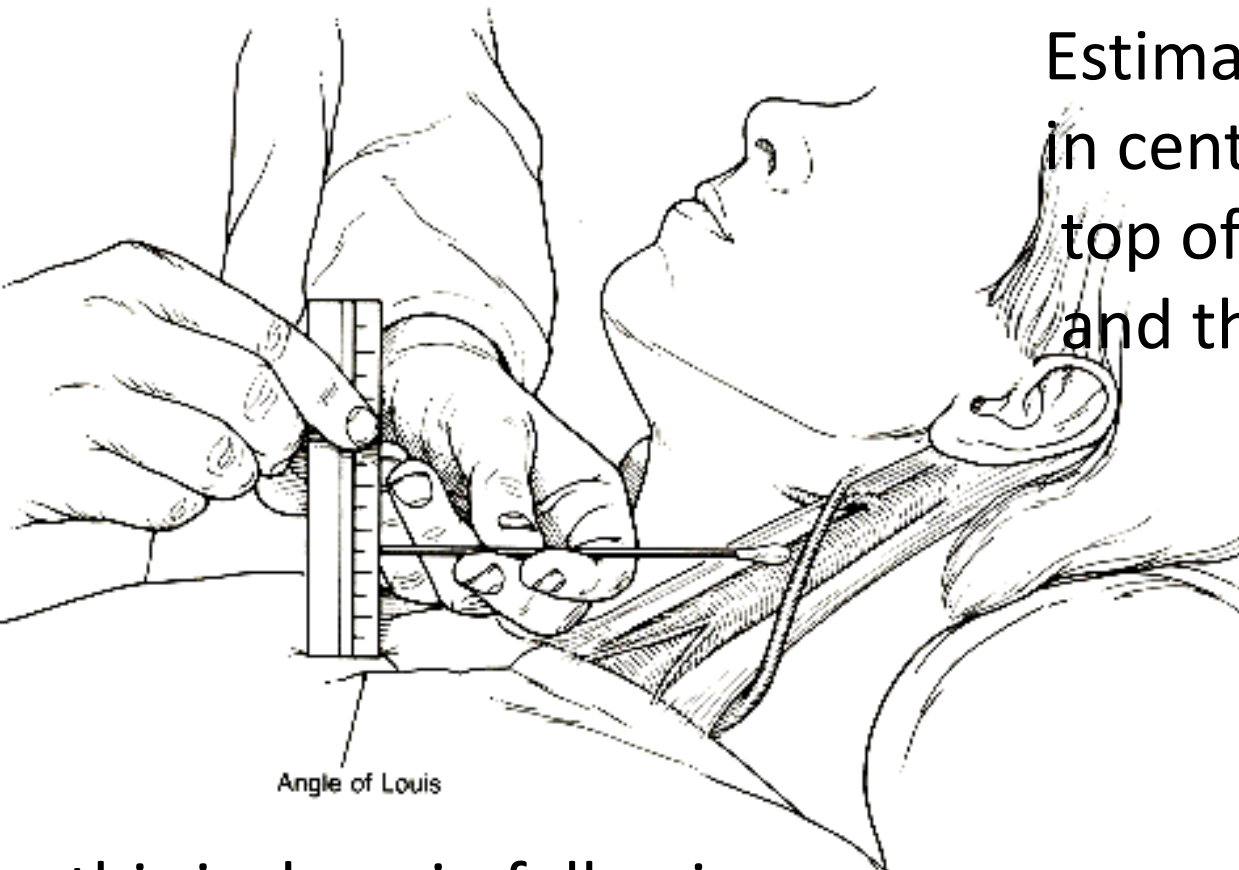
The clinician presses firmly over either the right upper quadrant of the abdomen (i.e., over the liver) or over the center of the abdomen for 10 seconds with a pressure of 20 to 35 mm Hg while observing the swelling of the internal jugular vein in the neck .

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Estimate the vertical height in centimetres between the top of the venous pulsation and the sternal angle

this is done in following way

keep one scale vertically on angle of louis and keep another scale horizontally at the highest point of venous pulsation in such a way that they will met with each other at  $90^{\circ}$

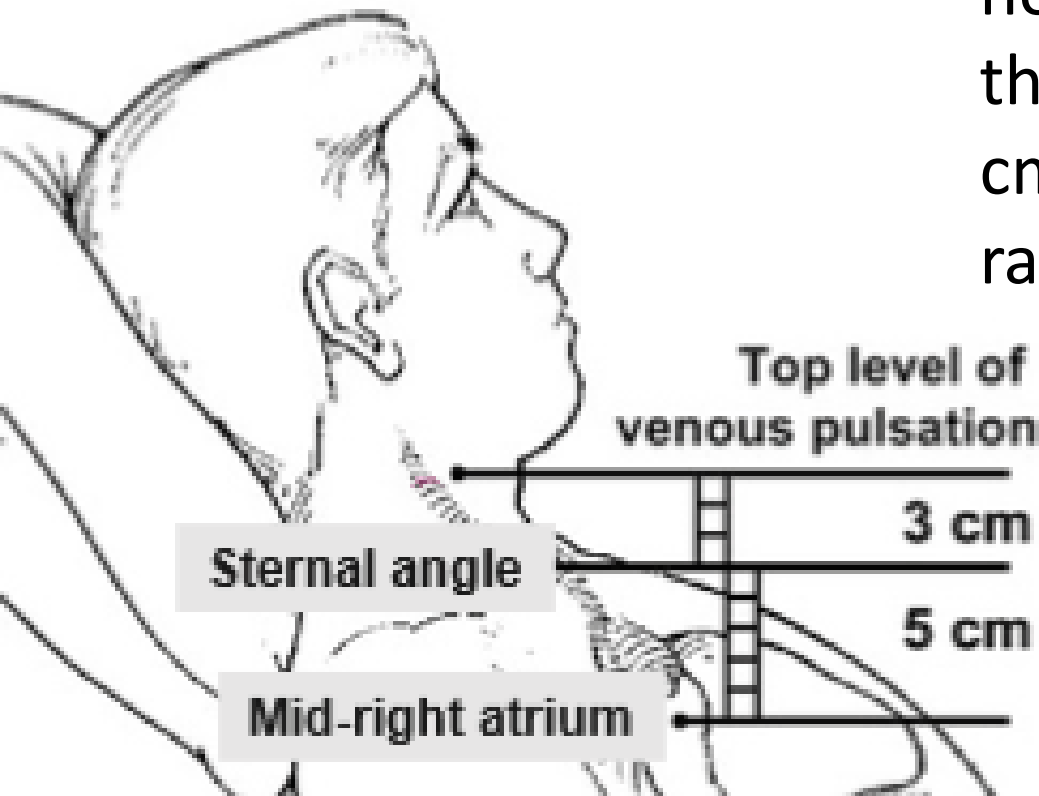
Now measure vertical height from angle of Louis to the point of intersect

3 cm (from sternal angle)  
+ 5 cm (from right atrium to sternal angle)  

---

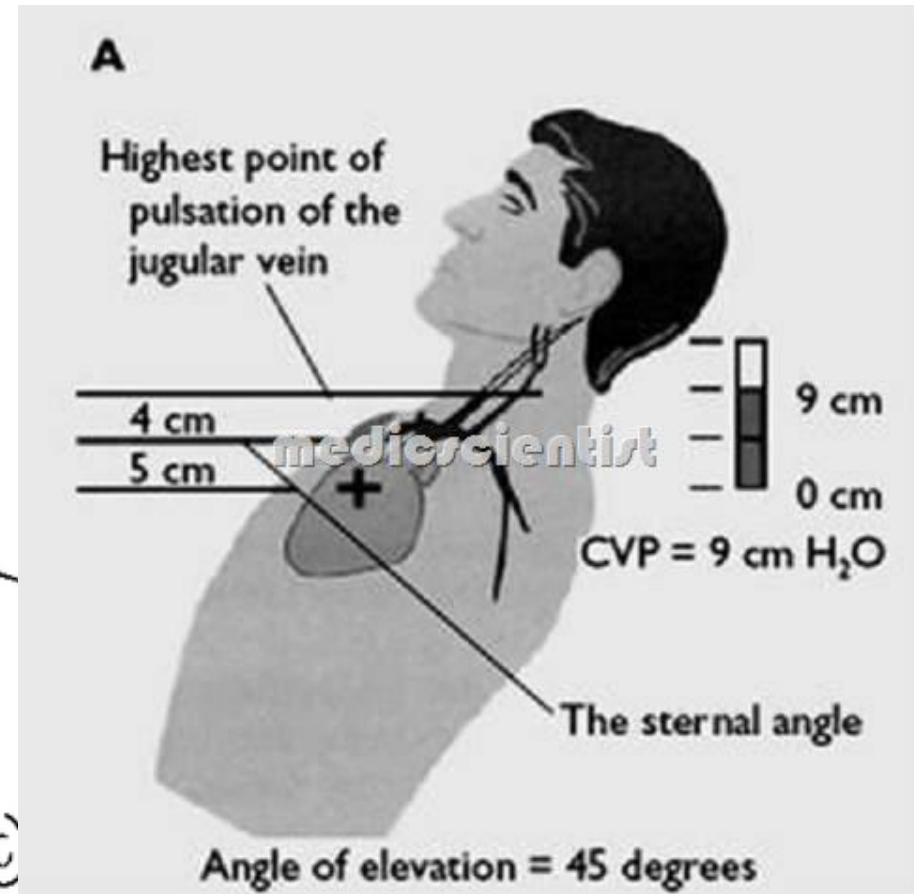
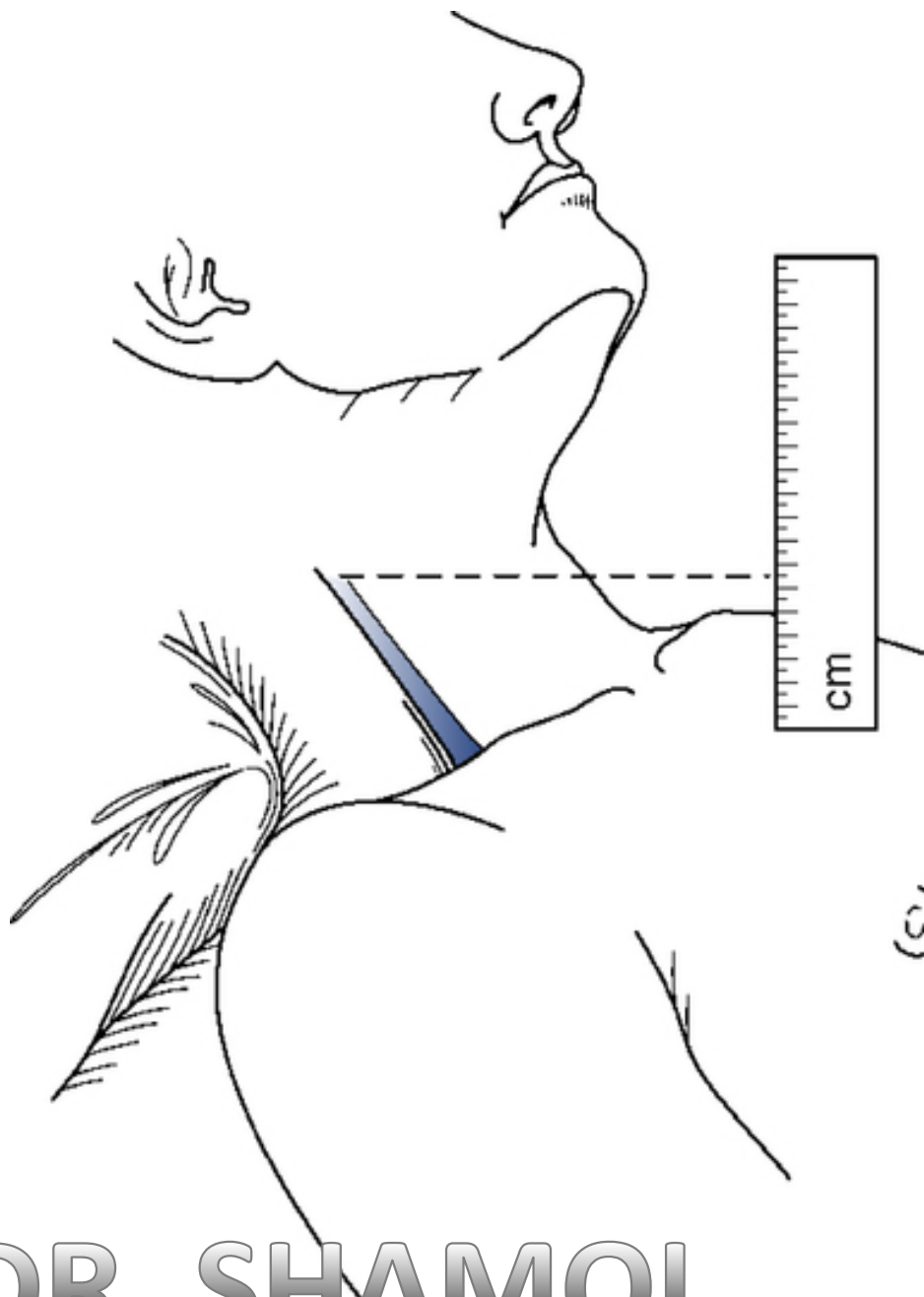
8 cm H<sub>2</sub>O jugular venous pressure

normally it will be not more than 4 cm if it is more than 4 cm than u will tell that JVP is raised



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Mean right atrial pressure is normally < 7 mmHg (9 cmH<sub>2</sub>O).  
Since the sternal angle is approximately 5 cm above the right atrium  
the normal jugular venous pulse should extend not more than 4 cm  
above the sternal angle



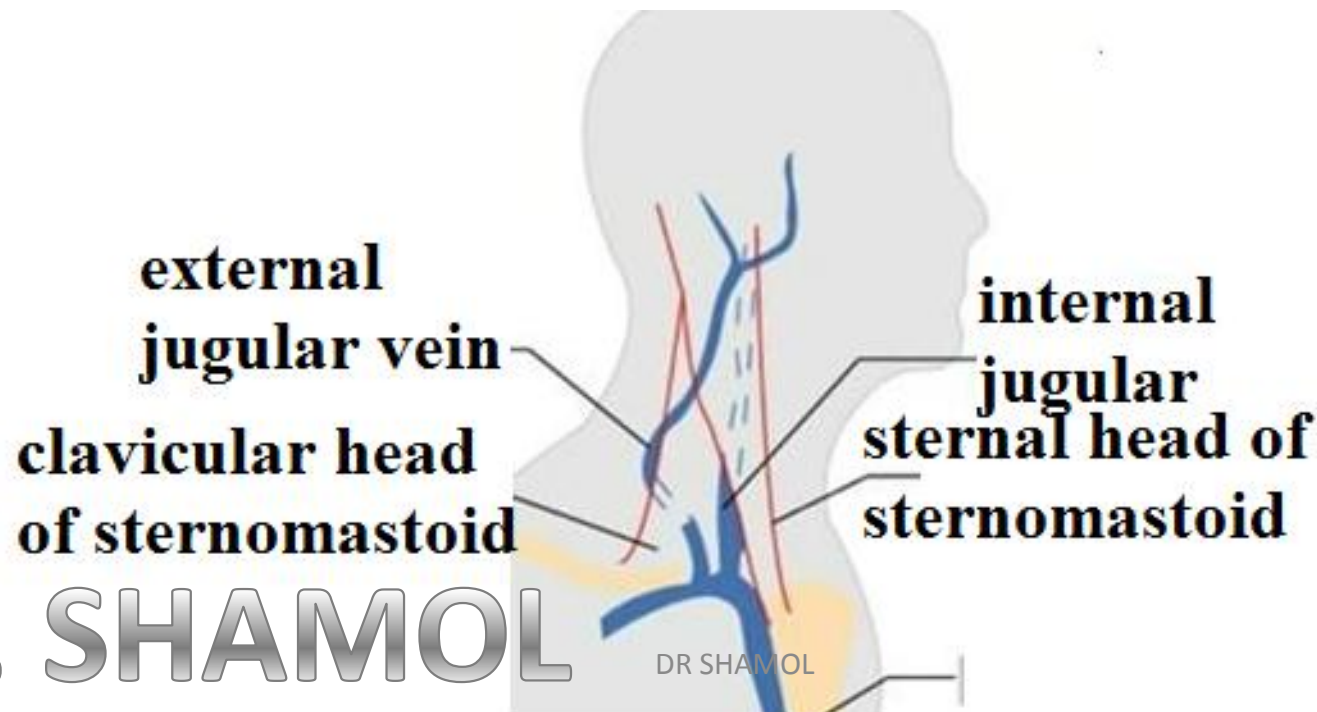
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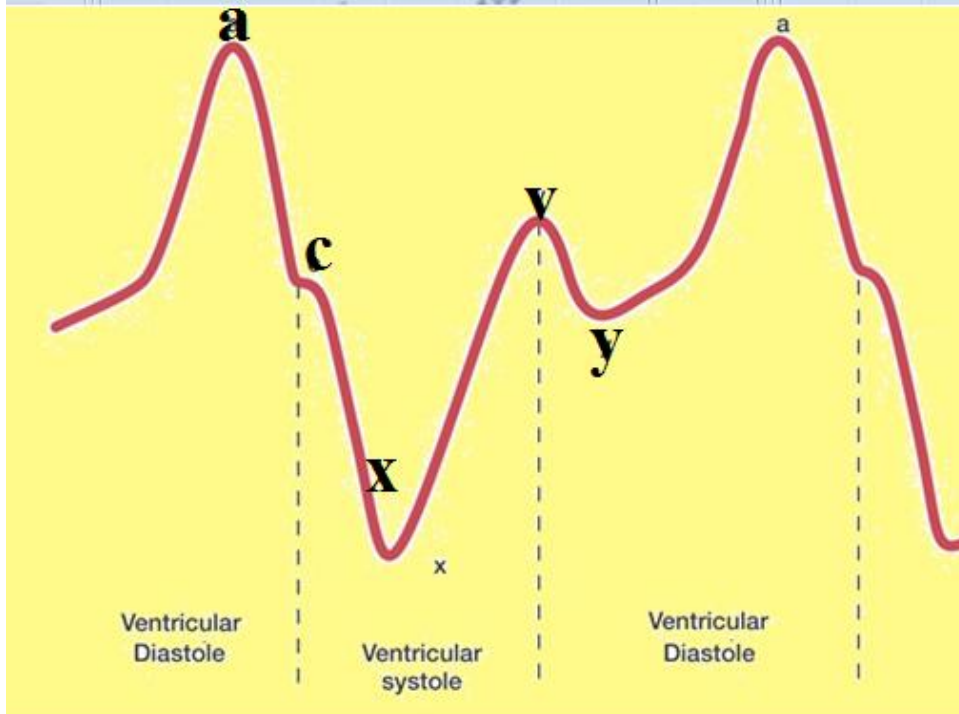


## **Why we see JVP in internal jugular vein than to external jugular vein?**

- There are no valves between the right atrium and the internal jugular vein. The degree of distension of this vein is therefore dictated by the right atrial pressure, and the venous waveform provides information
- The external jugular vein is more superficial and prominent. Do not examine this routinely because it is prone to kinking and partial obstruction as it traverses the deep fascia of the neck.

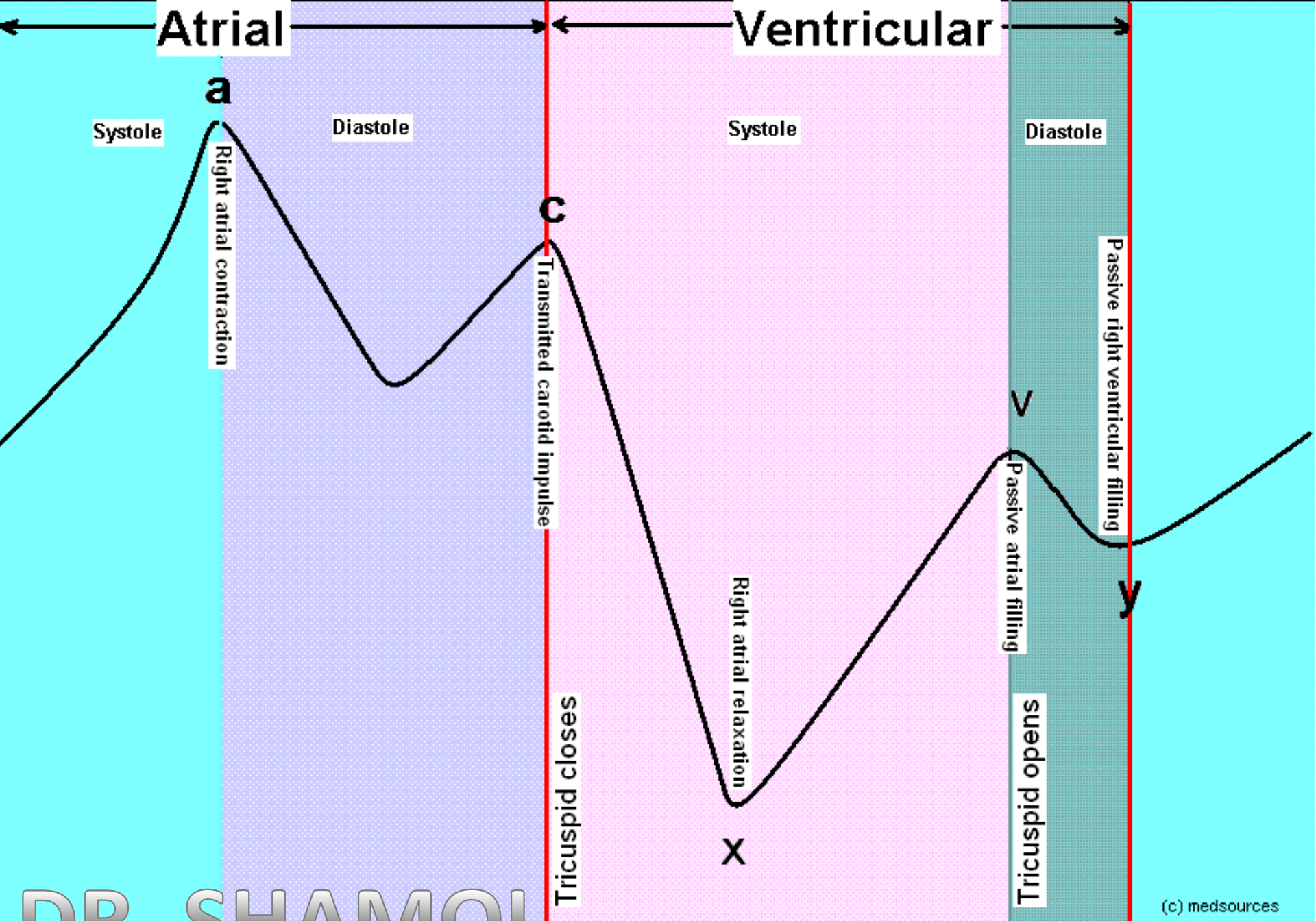


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" a " wave	corresponds to right <b>Atrial</b> contraction /systole
" c " wave	to trasmitted <b>Carotid</b> impulse or corresponds to right ventricular <b>Contraction</b> causing the triCuspid valve to bulge towards the right atrium.
The " x " descent	corresponds to atrial rela <b>Xation</b> and rapid atrial filling due to low pressure.
" v " wave	corresponds to <b>Venous</b> filling when the tricuspid valve is closed and venous pressure increases from venous return
" y " descent	corresponds to the rapid empt <b>Ying</b> of the atrium into the ventricle following the opening of the tricuspid valve.



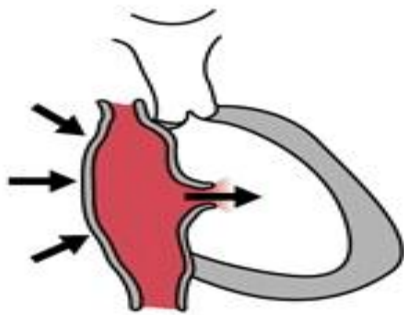
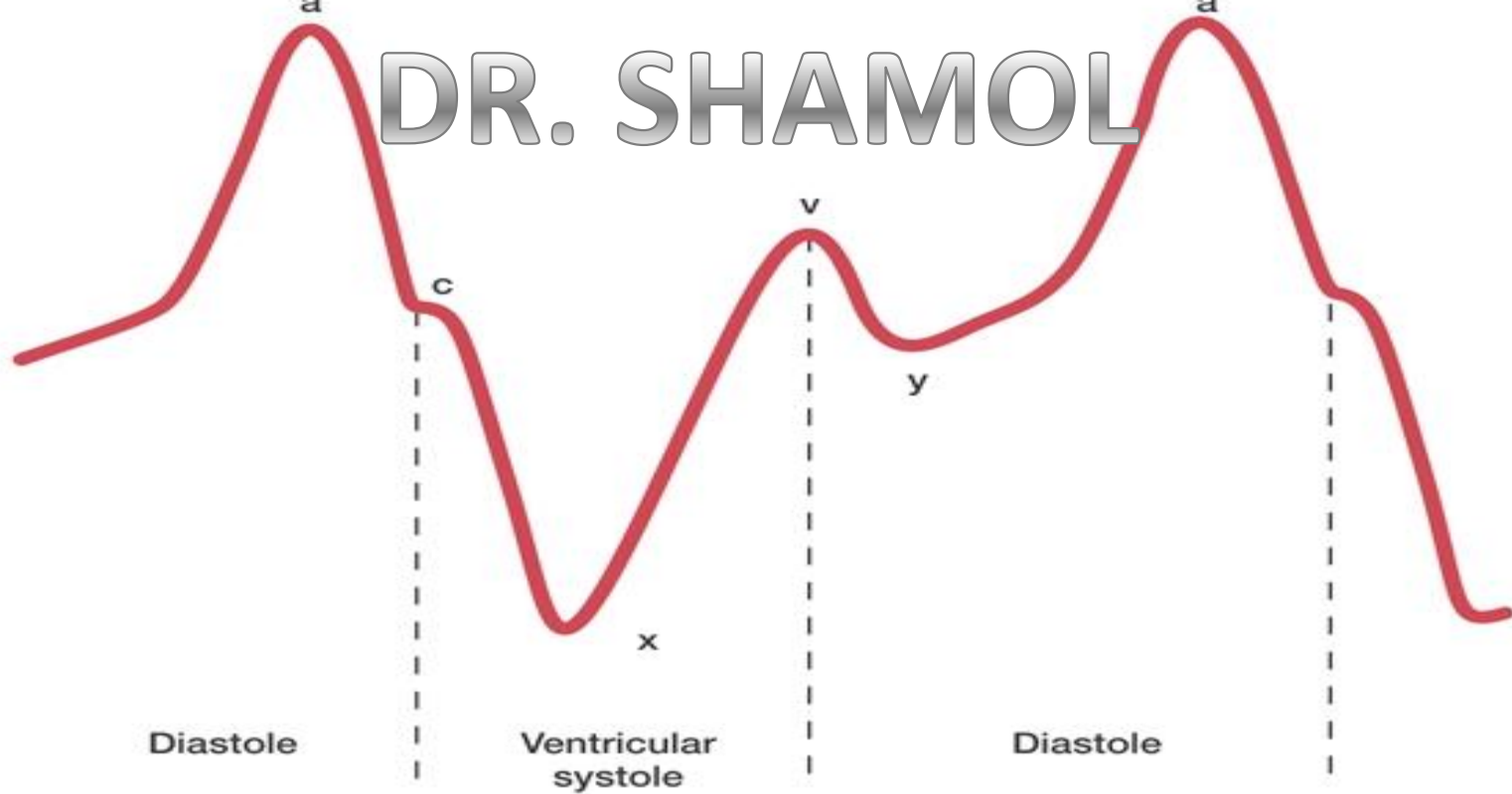
(c) medsources

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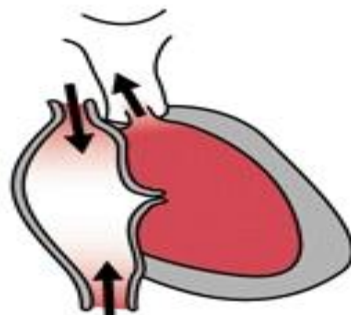


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**a wave**

Atrium contracting  
tricuspid valve open



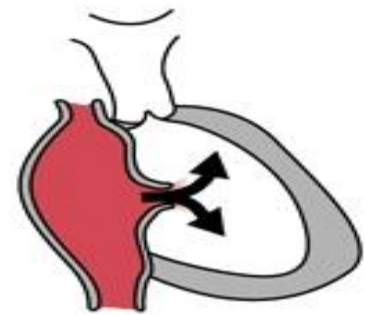
**x descent**

Atrium relaxing then  
filling, tricuspid  
closed



**v wave**

Atrium tense, full;  
tricuspid closed



**y descent**

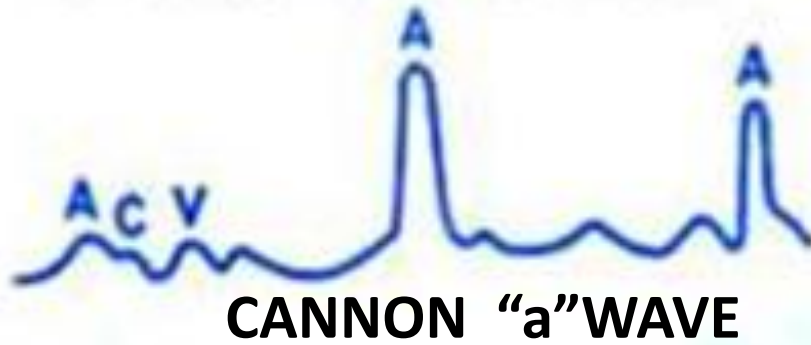
Atrium emptying,  
tricuspid open

## What are the causes of raised JVP

c	Congestive cardiac failure/ right heart failure	
	cor pulmonalae	
p	Pericardial effusion	
	Constrictive pericarditis	
	Pulmonary hypertension OR massive Pulmonary Embolism	
T	Tricuspid regurgitation	
	Tricuspid stenosis	
Non-pulsatile neck veins		superior venal caval obstruction

“ a” wave	Absent 'a' waves	Atrial fibrillation
	Giant 'a' waves	Tricuspid stenosis
	'Cannon' waves	Complete heart block
	Large wave	Pulmonary hypertension
Giant 'v' waves	Tricuspid regurgitation	
Steep ‘y’ wave	Constrictive pericarditis	
<b>Kussmaul’s sign</b>	↑JVP on inspiration	constrictive pericarditis
		cardiac tamponade

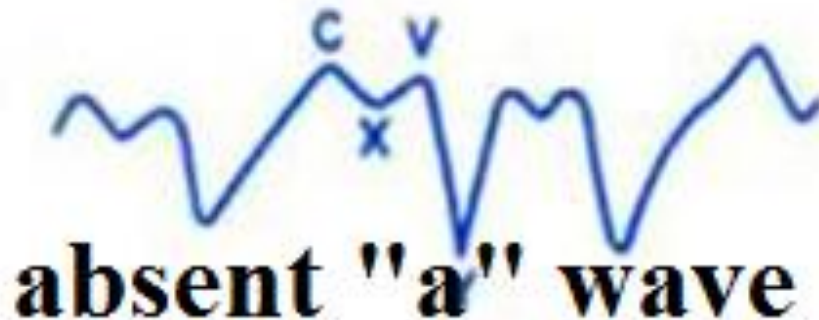
### G. Complete AV Block



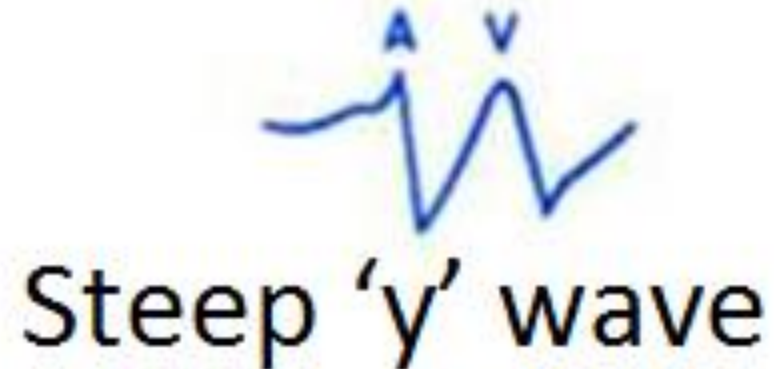
### B. Tricuspid Stenosis



### E. Atrial Fibrillation



### C. Constrictive Pericarditis



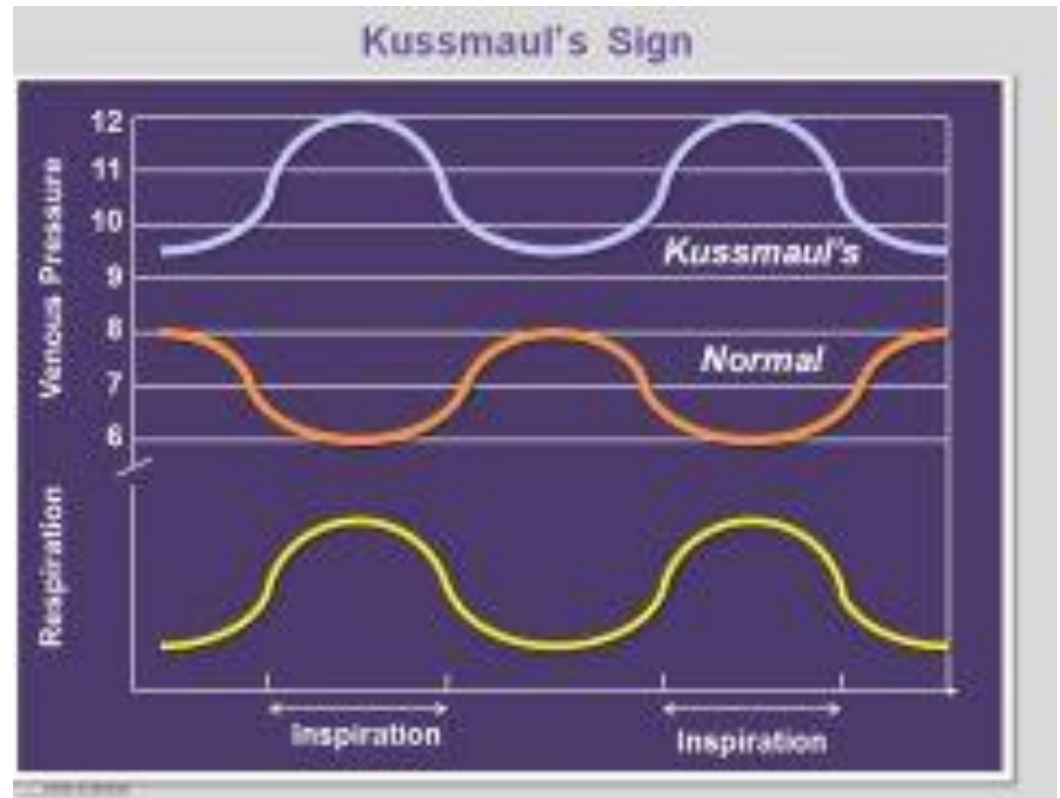


## Differences between the carotid artery and jugular vein pulsations

Carotid	Jugular
One pulsation per heartbeat	Double pulsation (a + v wave) per heartbeat (in sinus rhythm)
Rapid outward movement	Rapid inward movement
Palpable	Impalpable/ <b>visible</b>
Pulsation unaffected by pressure at the root of the neck	Obliteration of pulsation by pressure at root of the neck
Height independent of position of patient	Variation of height with position of patient
Independent of respiration	Healthy heart: ↓ JVP on inspiration Unhealthy heart (e.g. pericarditis): ↑ JVP on inspiration
Height unaffected with hepato-jugular reflux	Height increased with hepato-jugular reflux

## What is Kussmaul's sign?

It is paradoxically increased JVP in inspiration. Normally there is an inspiratory decrease in JVP. In constrictive pericarditis there is an inspiratory increase in JVP. Kussmaul's sign is also seen in severe right heart failure. It is caused by the inability of the heart to accept the increase in right ventricular volume without a marked increase in the filling pressure.





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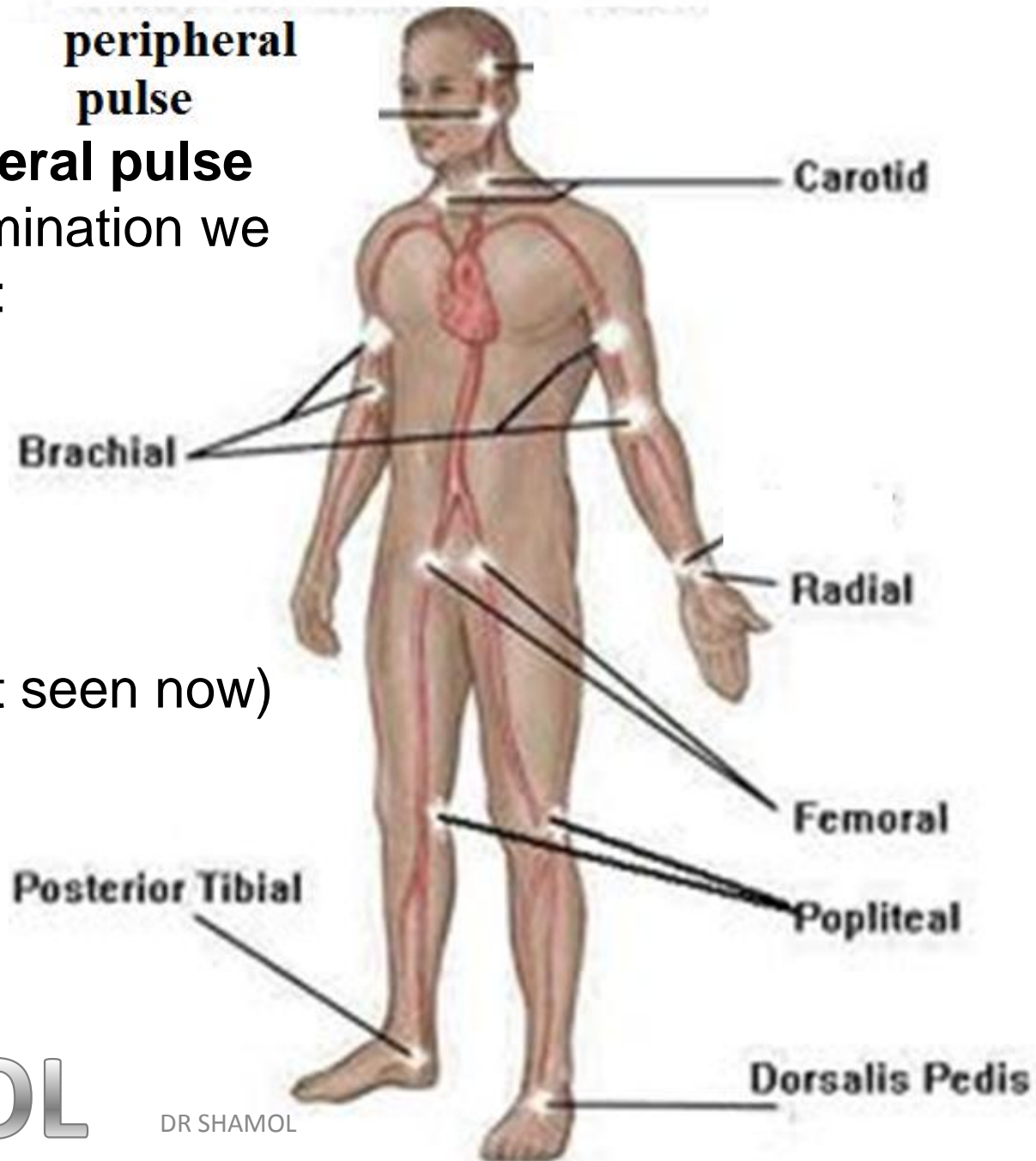
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## peripheral pulse

### Examination of peripheral pulse

In peripheral pulse examination we see the following pulse :

- Radial artery
- Brachial artery
- Carotid artery
- Femoral artery
- Popliteal artery
- Anterior tibial artery (not seen now)
- Posterior tibial artery
- Dorsalis pedis





Examination of

Radial pulse

The radial pulse is felt on the wrist, just under the thumb



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- ❖ hand shake with patient with the right hand
- ❖ if u do it the hand will automatically remain in semiprone and semiflex position
- ❖ Place your three middle fingers over the right radial pulse.
  - ❖ ring finger will regulate the pulse
  - ❖ middle finger will feel the pulse
  - ❖ index will prevent retrograde pulsation
- ❖ Count the pulse for 15 seconds and multiply by four to obtain the pulse rate in beats per minute
- ❖ The *radial pulse* is found at the wrist, lateral to the flexor carpi radialis tendon and medial to the radial styloid process at the wrist.



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index finger will prevent  
retrograde pulsation

middle finger will feel the  
pulse

ring finger will regulate the  
pulse

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index finger will prevent  
retrograde pulsation

middle finger will feel the  
pulse

ring finger will regulate the  
pulse

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# Radio-radial and Radio-femoral delay



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To see  
radio-radial  
delay

Now palpate the left radial pulse with right three finger simultaneously keeping the left hand on right radial pulse to detect any radio-radial delay present or not





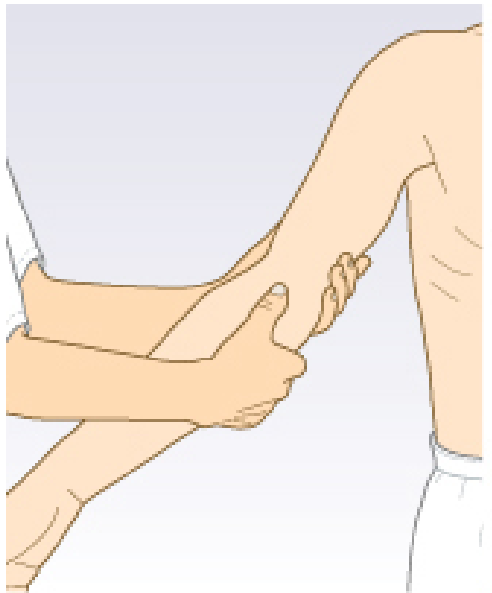
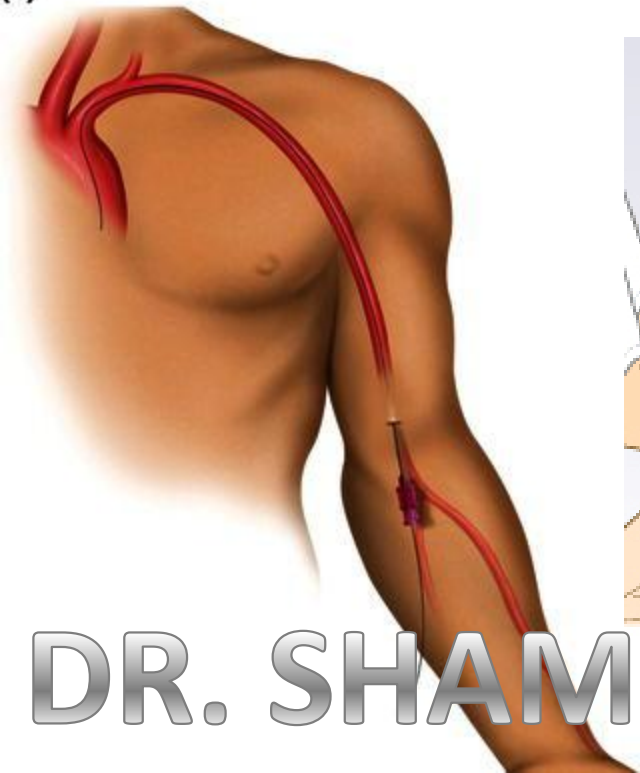
To see radio-femoral delay

Now place right hand just below the mid inguinal region to see femoral pulse and simultaneously keeping the left hand on right radial pulse

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# Examination of Brachial pulse

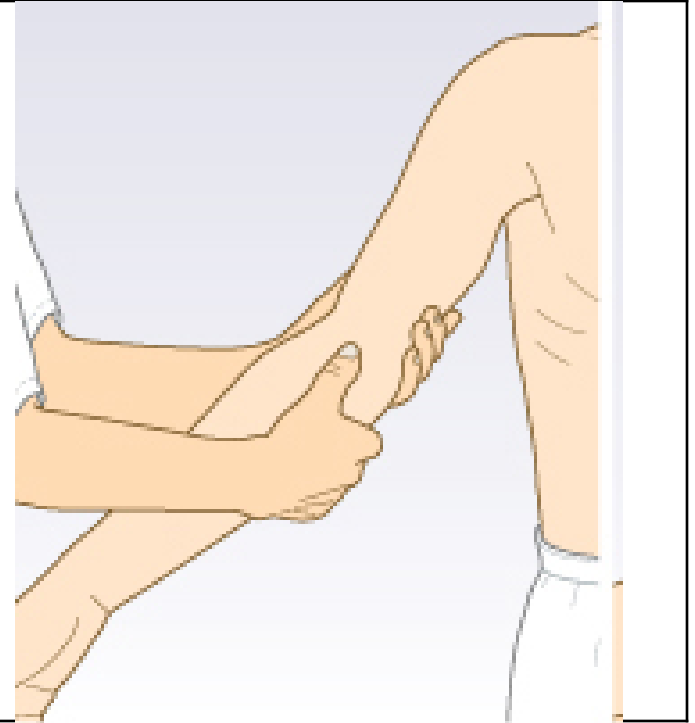
(a)



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1. Use your thumb (right thumb for right arm and vice versa) with your fingers cupped round the back of the elbow
2. Feel medial to the tendon of the biceps muscle to find the pulse and assess its character.
3. Feel medial to the tendon of the biceps muscle to find the pulse and assess its character.







First semi flex the elbow to make biceps tendon prominent

Identify or feel the tendon with thumb



Now place the left thumb medial to right tendon and feel the brachial artery



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Examination of

Carotid pulse



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1. Never compress both carotid arteries simultaneously.
2. Use your left thumb for the right carotid pulse and vice versa.
3. when you see the right carotid pulse ask the patient to turn head toward the left
4. Place the tip of your thumb between the larynx and the anterior border of the sternocleidomastoid muscle.
5. Press your thumb gently backwards to feel the pulse







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left thumb for  
the right carotid pulse

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Right thumb for the  
left carotid pulse

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Alternately you can see  
with two finger also

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## PULSE OF LOWER LIMB



Femoral pulse



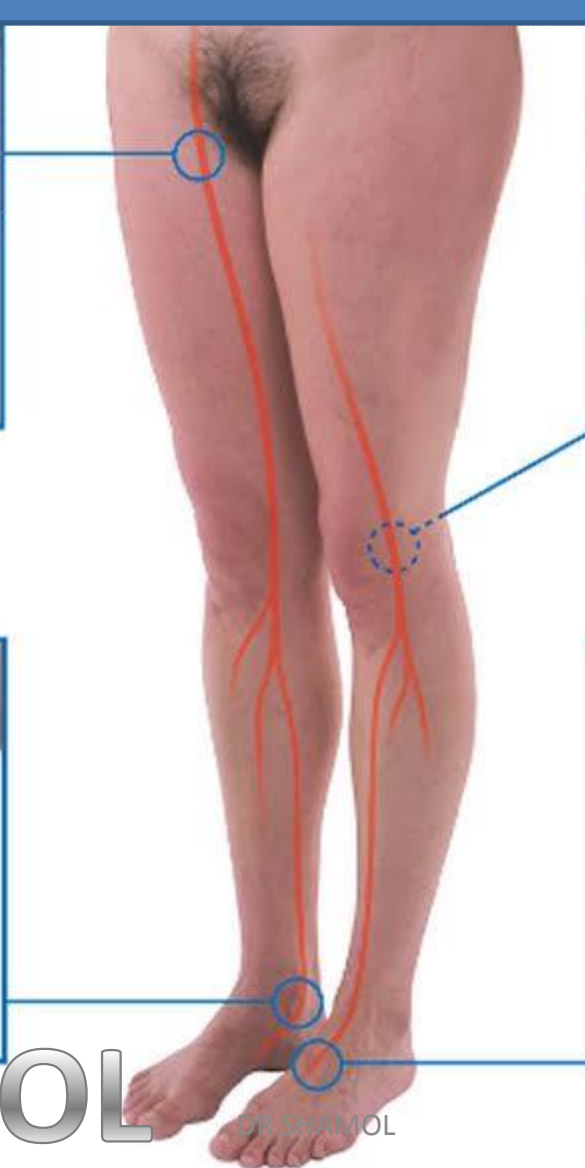
Popliteal pulse



Posterior tibial pulse



Dorsalis pedis pulse



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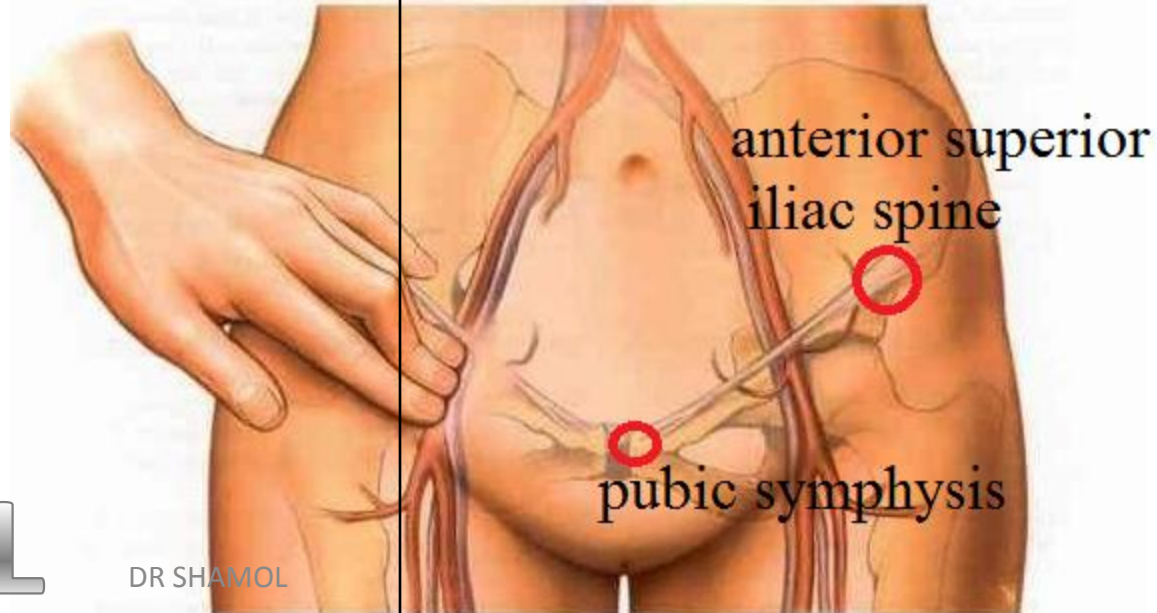


Pulse in lower limb

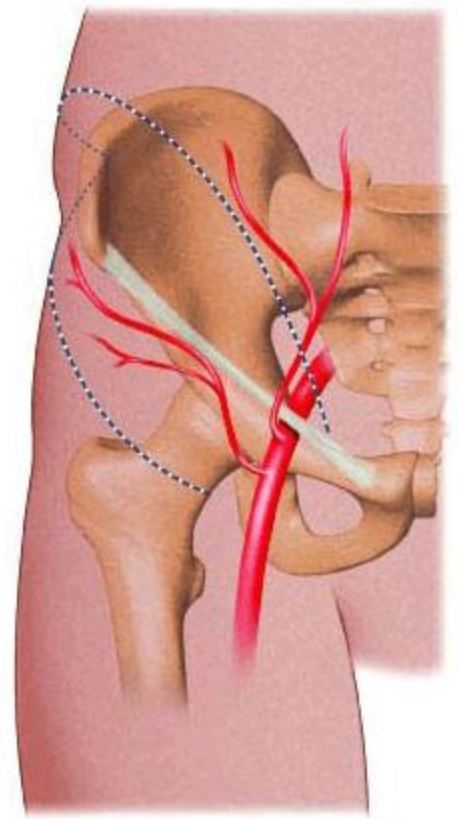
Femoral pulse

## Location of femoral pulse

1. The *femoral artery* is situated just below the inguinal ligament at the mid-inguinal point which is midway between the anterior superior iliac spine and the pubic symphysis
2. The pt will be in supine position firmly press downwards at mid inguinal point using two or three extended fingers.



First identify anterior superior iliac spine and the pubic symphysis  
In our country do it above the clothes



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firmly press downwards at mid inguinal point using two or three extended fingers.

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Now feel the left femoral pulse in same way



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Examination

## Popliteal pulse



1. The patient should lie on a firm comfortable surface so they can relax their muscles.
2. Flex the patient's knee to 30°.
3. Keep your both thumbs in front of the patients knee and rest of your fingers behind popliteal fossa
4. Press firmly in the midline with your finger s over the popliteal artery.
5. By sliding your fingers 2-3 cm below the knee crease it may be possible to compress the artery against the back of the tibia







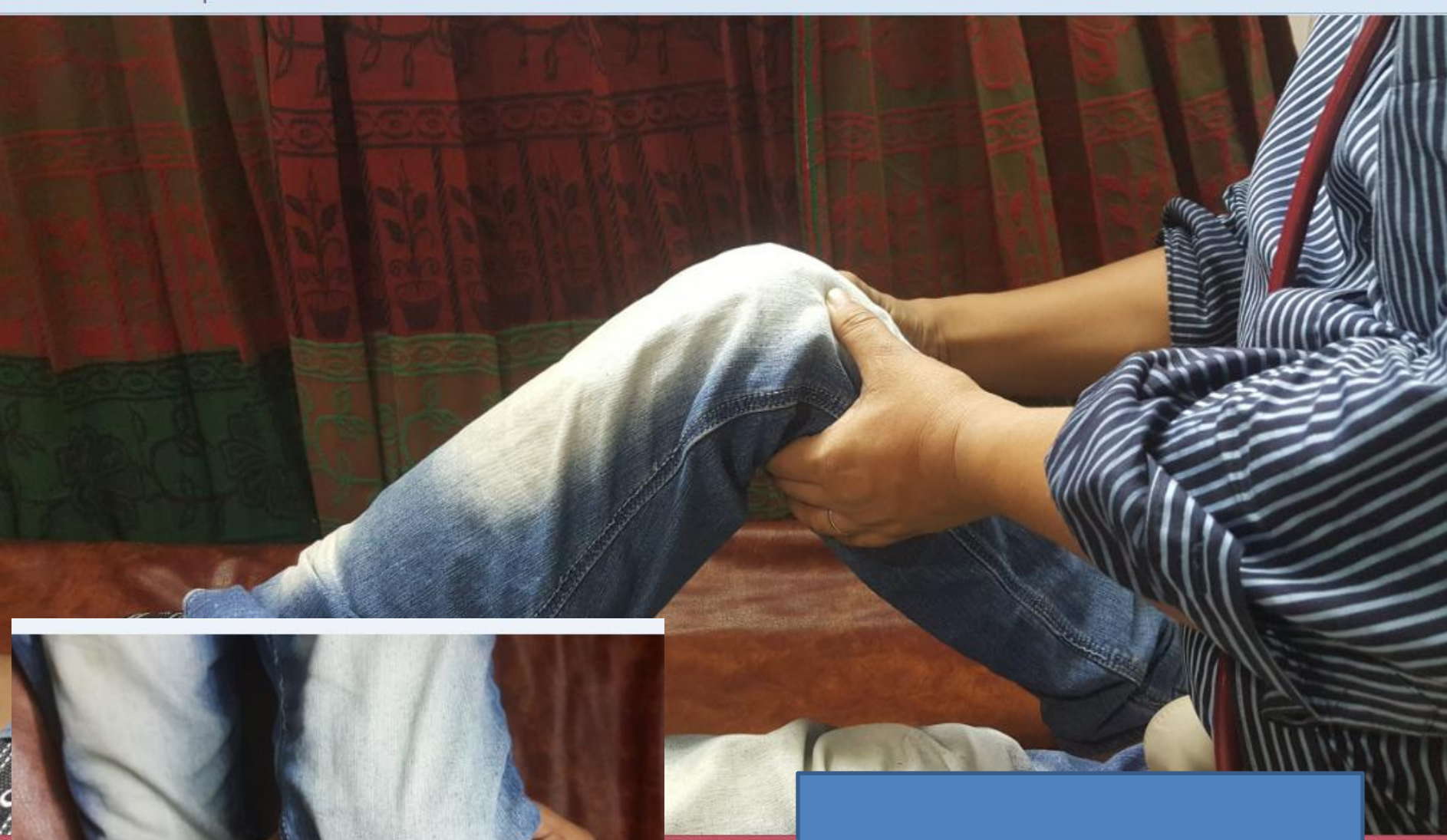
Right Popliteal pulse

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left Popliteal pulse



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# Examination of

## Posterior tibial artery

Medial malleolus

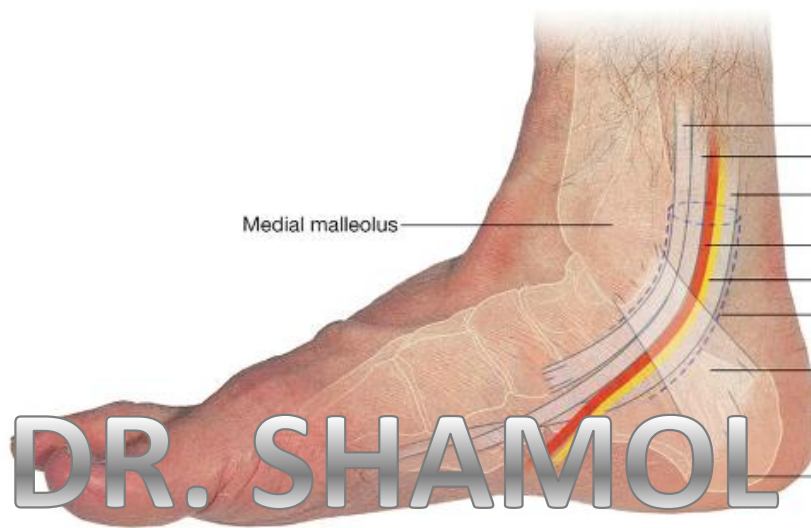
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Medial malleolus

Posterior tibial artery

Calcaneal tendon



1. Patient will b in lying position
2. place your index and middle fingers 2 cm below and 2 cm behind the medial malleolus & press against the bone to feel the pulse





place your index and middle fingers 2 cm below and 2 cm behind the medial malleolus & press against the bone to feel the pulse



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You can see each pulse separately

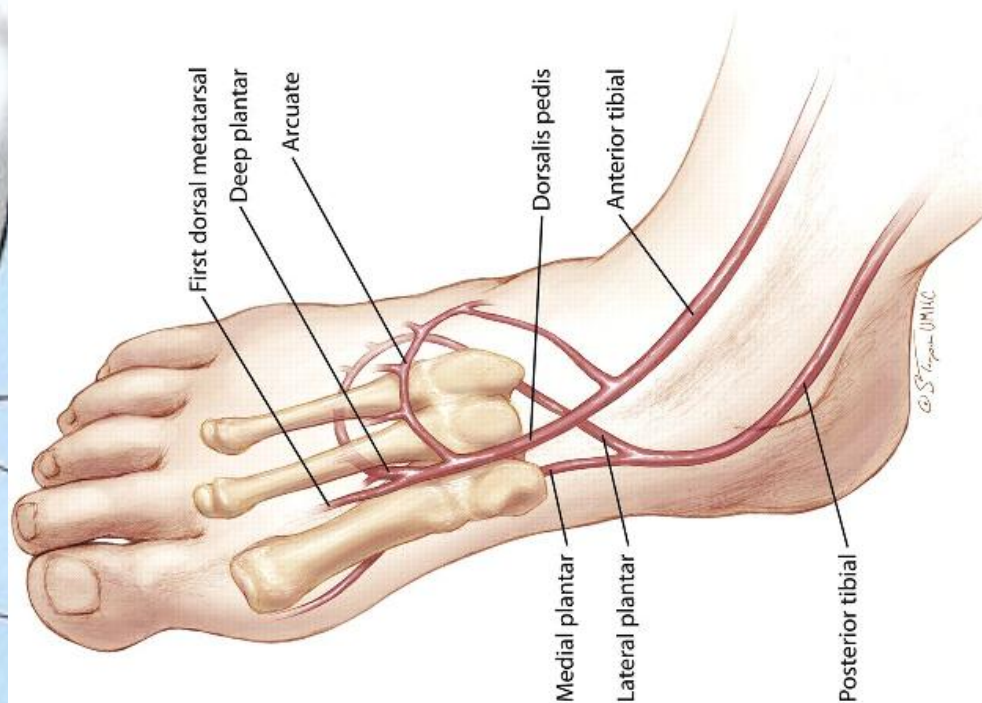
Or like this both right and left post. Tibial pulse at a time

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# Examination

## arteries dorsalis pedis



Tibialis anterior  
tendon

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1. The *dorsalis pedis artery* is the continuation of the anterior tibial artery on the dorsum of the foot
2. It passes lateral to the tendon of extensor hallucis longus and is best felt at the proximal extent of the groove between the first and second metatarsals
3. First place index , middle and ring finger in the middle of the dorsum foot lateral to the tendon of extensor hallucis longus .
4. Be careful that your fingers should remain in the grooves between the first and second metatarsals
5. if u still cannot find the or feel the pulse please extend the great toe against resistance . it will make the tendon prominent & now feel the pu





please extend the great toe against resistance . it will make the tendon prominent

Now place index , middle and ring finger in the middle of the dorsum foot lateral to the tendon of extensor hallucis longus .





Now see left  
arteries dorsalis pedis  
In same way







You may also see two  
artery at a time

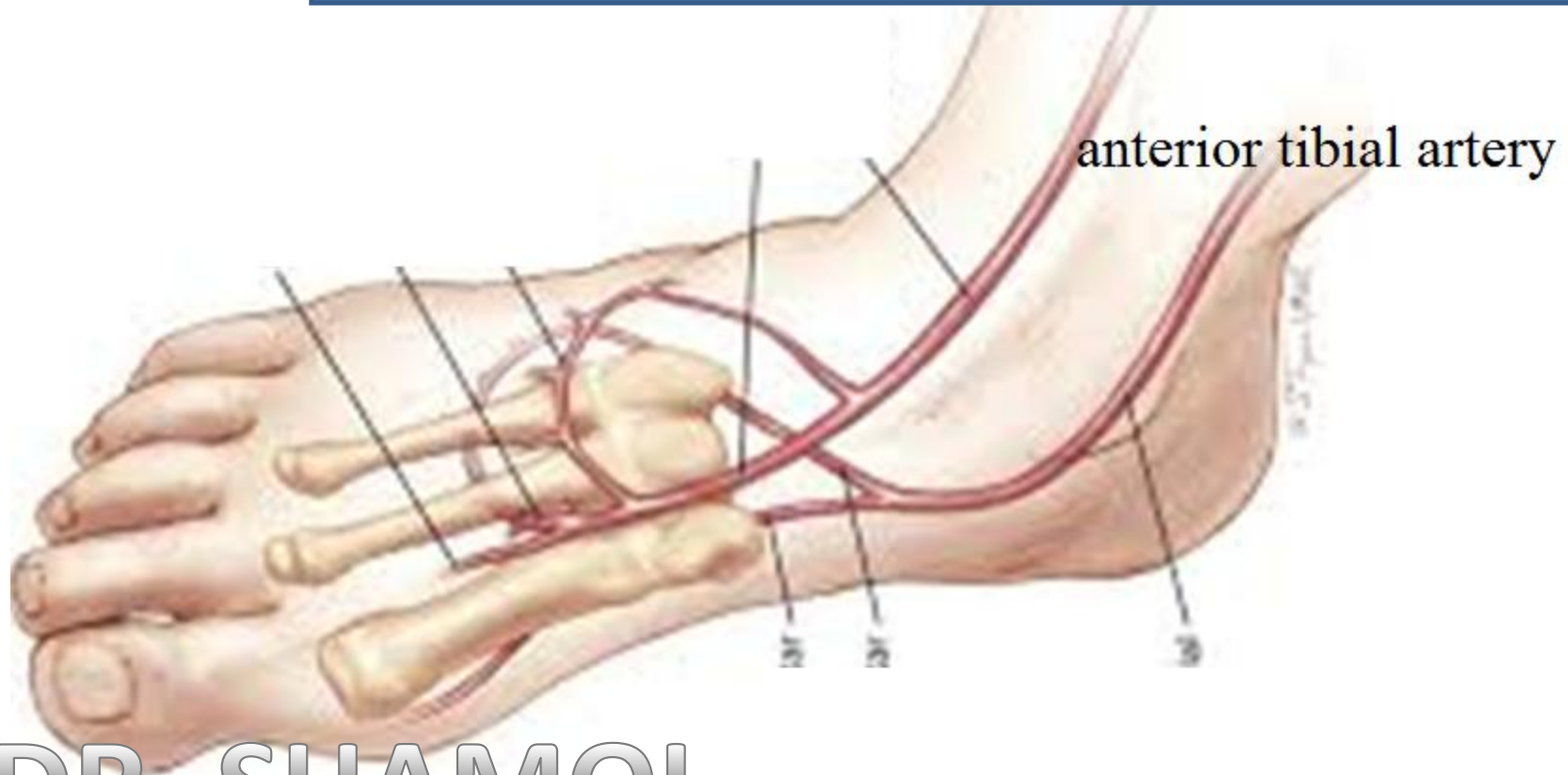
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Examination  
Not done

Anterior tibial artery



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1. Patient will b in lying position
2. Place your index and middle fingers in between medial and lateral malleolus
3. Now try to feel the pulse

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1. Patient should be in lying position
2. Ask the patient if there any pain in the elbow or shoulder joint.
3. Now grasp the right hand of the patient with your left hand
4. Now with your right hand grasp right forearm just below wrist joint in such a position that the ball of the right finger will remain over remain over the radial pulse
5. Now fell the pulse with the ball of the right finger for few second
6. Then suddenly raised the patient 's arm above the head or heart level
7. Now feel the pulse for few second







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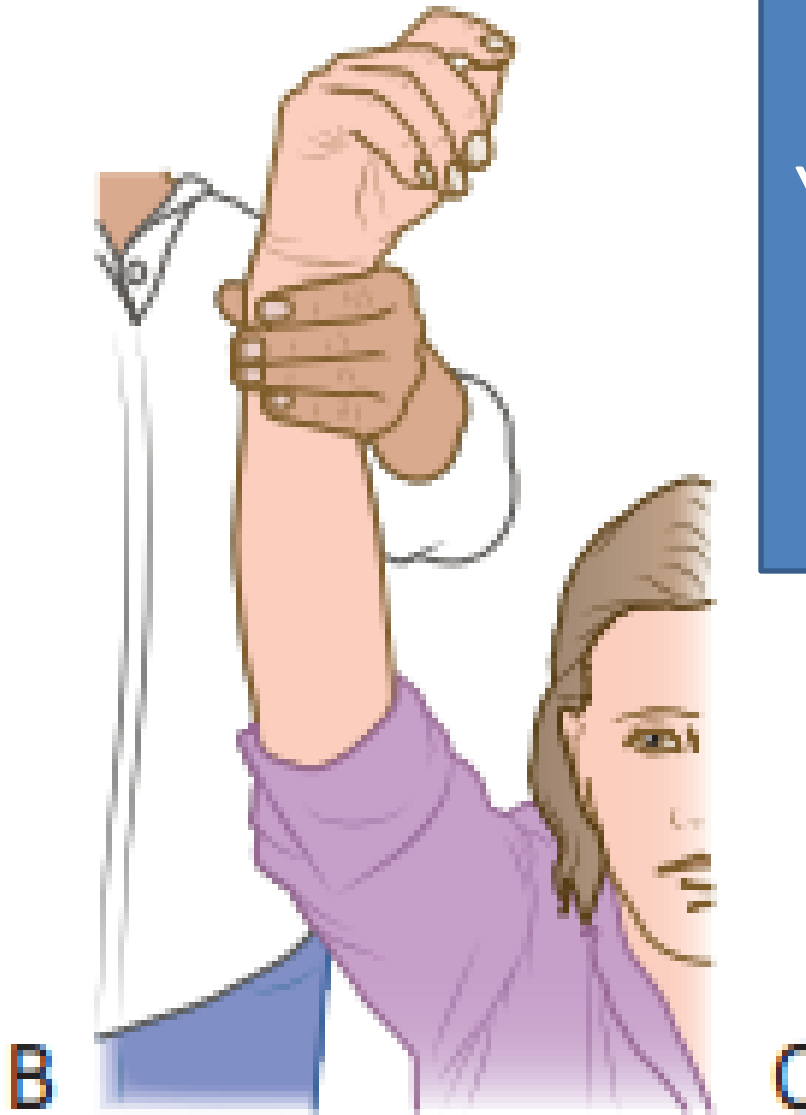


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YOU MAY SEE WITH SINGLE  
HAND



achial and carotid pulse

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WHAT  
WILL  
I LOOK  
IN PULSE

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## **In examination of pulse what will u see?**

Rate

Rhythm

Volume

Character

Radio-radial delay

Radio-femoral delay

Condition of vessel wall

Rate and rhythm seen –radial artery

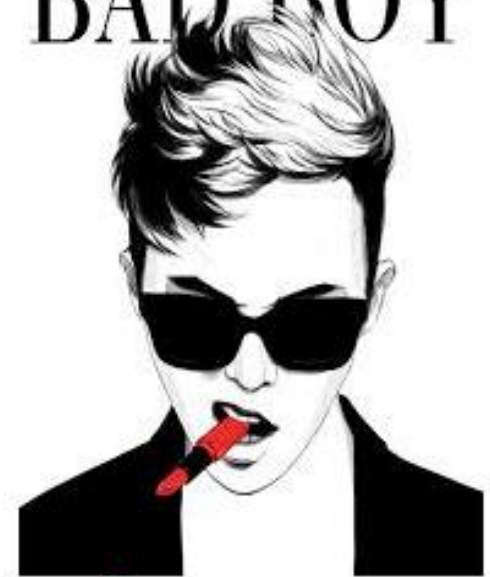
Volume and character is seen in carotid artery  
also in brachial & femoral artery



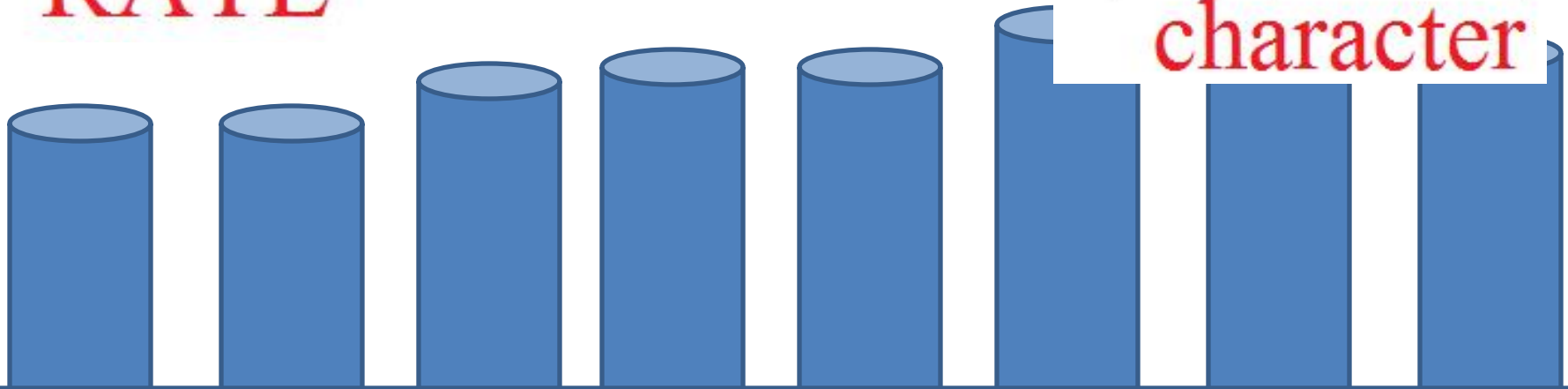


RATE

BAD BOY

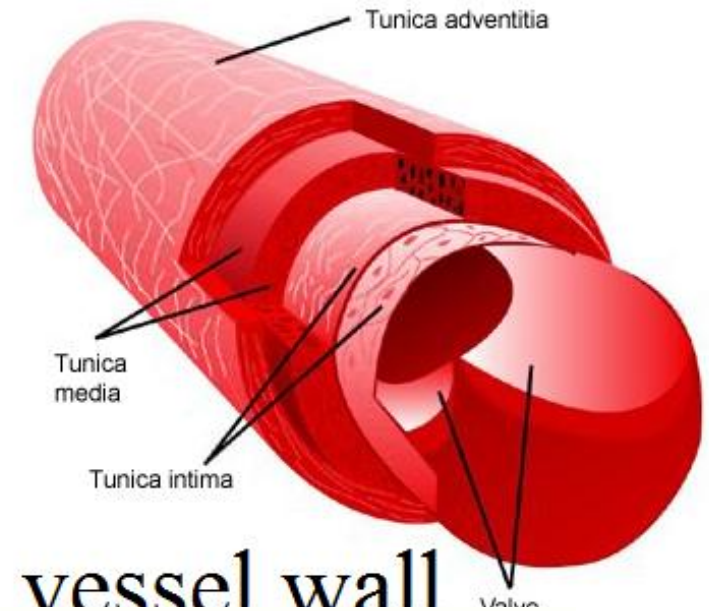
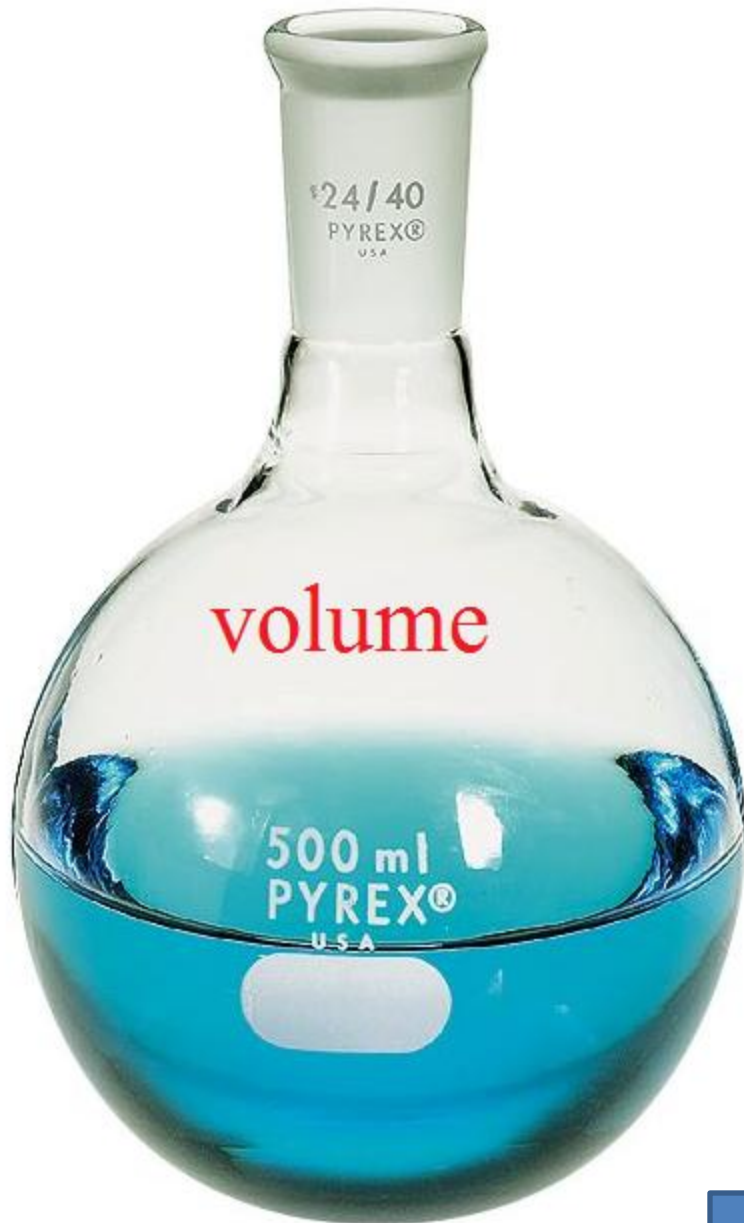


character



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## QUESTION REGARDING PULSE



sinus tachycardia



sinus bradycardia

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## Causes or sinus tachycardia and sinus bradycardia ?

### Cause of sinus tachycardia

#### Fast heart rate (tachycardia, > 100/min)

- **Physiological**
  - Exercise
  - Pain
  - Excitement/anxiety
- Hyper dynamic circulation
  - Fever
  - Hyperthyroidism
- Medication:
  - sympathomimetics  
sulbutamol
  - vasodilators
- **Pathological**
  - Atrial fibrillation
  - Atrial flutter
  - Supraventricular tachycardia

Ventricular tachycardia

### Cause of sinus bradycardia?

#### Slow heart rate (bradycardia, < 60/min)

- **Sinus bradycardia**
  - Sleep
  - Athletic training
  - Hypothyroidism
  - Medication:
    - Beta-blockers
    - Digoxin
    - Verapamil, diltiazem
- **Pathological**
  - Carotid sinus hypersensitivity
  - Sick sinus syndrome
  - Second-degree heart block
  - Complete

<b>What are causes of irregular pulse?</b> <ul style="list-style-type: none"> <li>• Irregularly irregular <ul style="list-style-type: none"> <li>○ Atrial fibrillation</li> <li>○ Atrial flutter with variable response</li> <li>○ Multiple ectopics</li> </ul> </li> <li>• Regularly Irregular <ul style="list-style-type: none"> <li>○ Sinus arrhythmia</li> <li>○ Second-degree heart block Type –I</li> <li>○ Ventricular extrasystoles</li> </ul> </li> </ul>	<b>Cause of low volume pulse?</b> <ul style="list-style-type: none"> <li>• Shock</li> <li>• Aortic stenosis</li> <li>• Pericardial effusion</li> <li>• Pulmonary hypertension</li> </ul>
<b>Causes of radio radial delay and radio-femoral delay?</b> <ul style="list-style-type: none"> <li>• Radio-femoral delay <ul style="list-style-type: none"> <li>○ coarctation of the aorta distal to left subclavian artery</li> </ul> </li> <li>• Radio-radial delay <ul style="list-style-type: none"> <li>○ coarctation of aorta proximal</li> </ul> </li> </ul>	<b>Cause of high volume ?</b> <ul style="list-style-type: none"> <li>• AR</li> <li>• Hyperdynamic circulation <ul style="list-style-type: none"> <li>○ Fever</li> <li>○ Pregnancy and</li> <li>○ Thyrotoxicosis</li> <li>○ PDA</li> </ul> </li> </ul>
<b>what are causes of absence of pulse in upper limb ?</b> <ul style="list-style-type: none"> <li>• takayasu disease</li> <li>• Atherosclerosis</li> <li>• thrombo-embolism</li> <li>• aberrant vessel</li> </ul>	<b>causes of absence of pulse in lower limb ?</b> <ul style="list-style-type: none"> <li>• peripheral arterial diseases</li> <li>• Buerger's disease (thromboangiitis obliterans)</li> <li>• Vasculitis</li> </ul>

**What are feature of coarctation of aorta ?**

- patient have headache
- pulse –radio-femoral delay
- BP—more in upper limb than in lower limb
- Murmur ---systolic murmur at midscapular region

**What are feature of takayasu disease ?**

**it is vasculitis**

- patient have HO claudication in upper limb
- pulse ---absent in upper limb
- BP—high
- bruit ---renal , carotid present
- heart –Murmur of aortic regurgitation





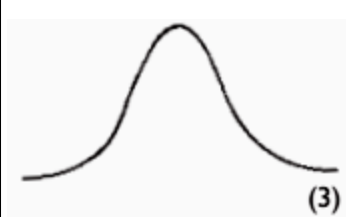
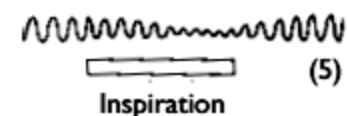


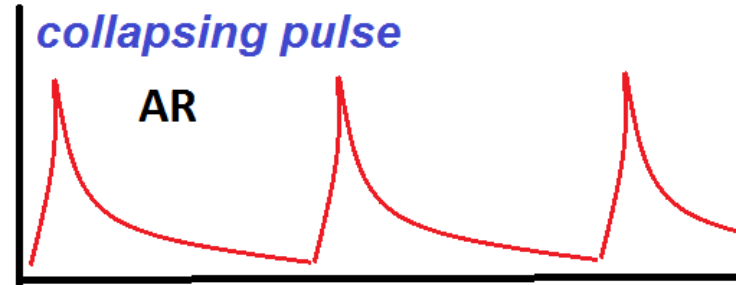
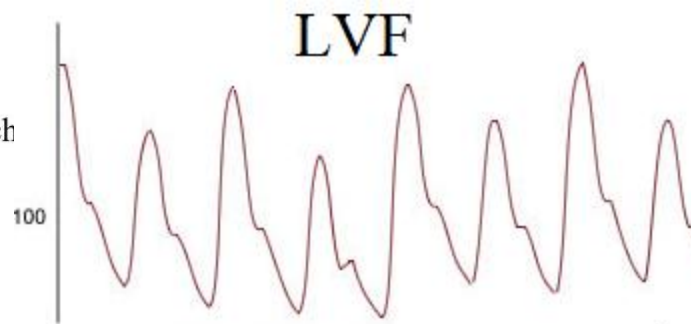
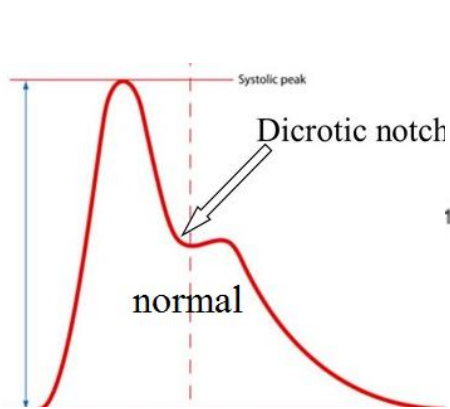
# CHARACTER OF PULSE



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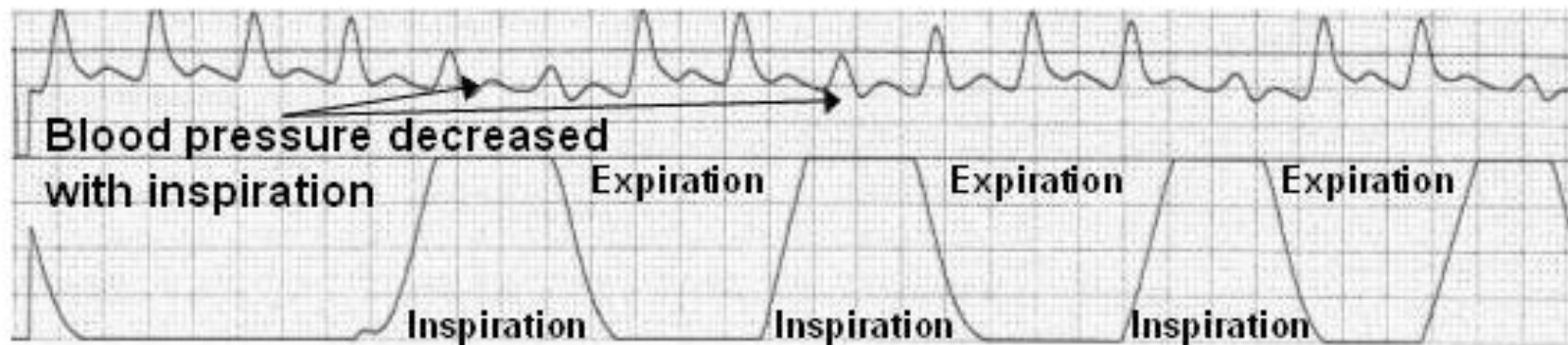
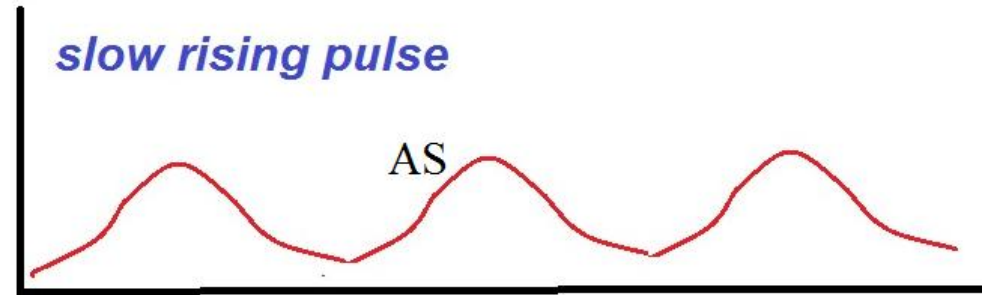
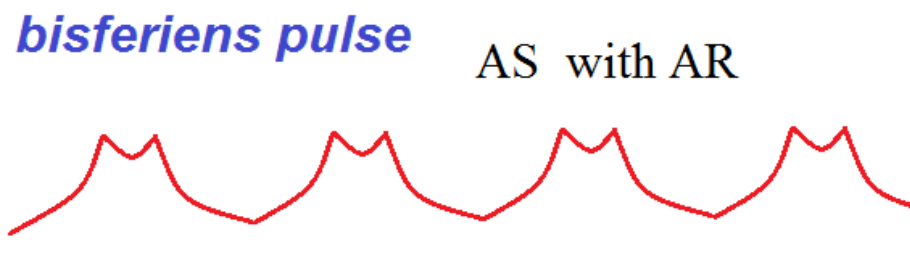
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type of different pulse				
	normal			(1)
A	Anacrotic	Slowly rising & small volume pulse	Aortic stenosis	
A	Pulsus alternans	an alternating strong and weak pulsation	LVF	
B	Pulsus bisferiens	Double peak of pulse , combination of slow rising and collapsing pulse	AS with AR	
C	collapsing	Rapid upstroke and descend of pulse. the pulse which feels as though it suddenly hits your fingers and falls away just as quickly and seen by raising the arm above the head .	<ul style="list-style-type: none"> <li>• AR,</li> <li>• Hyperdynamic circulation</li> <li>• PDA</li> <li>• Rupture of sinus of Valsava</li> <li>• Large A-V fistula</li> </ul>	
W	waterhammer	Collapsing pulse of AR is called water hammer pulse	AR	
P	Pulsus paradoxus	When volume of pulse reduce in inspiration and increase in expiration then it is called pulsus paradoxus .it is the exaggeration of normal phenomenon .	<ul style="list-style-type: none"> <li>• Pericardial effusion</li> <li>• Chr.constrictive pericarditis</li> <li>• Acute severe asthma</li> <li>• Massive pulmonary embolism</li> </ul>	
J	Jerky pulse	:	Hypertrophic cardiomyopathy	

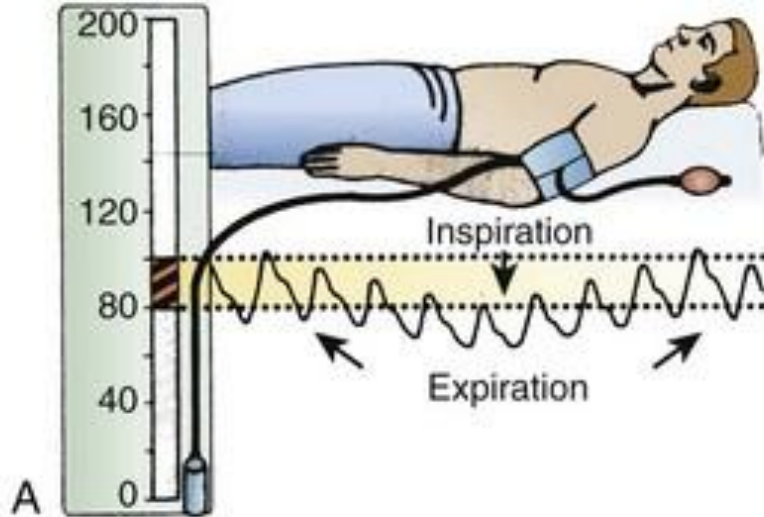


Pulsus alternans

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pericardial effusion, sever asthma



### PROCEDURE FOR THE MEASUREMENT OF PULSUS PARADOXUS

The patient should be reclining at a 30° to 45° angle and instructed to breathe normally.

1. Inflate a standard blood pressure cuff until Korotkoff sounds over the brachial artery disappear.
2. Lower pressure in the cuff a few millimeters of mercury per second until the first Korotkoff sounds appear during expiration.
3. Maintain pressure at this level and observe the disappearance of sounds during inspiration. Record this cuff pressure.
4. Very slowly lower cuff pressure until Korotkoff sounds are heard throughout the respiratory cycle. Record this cuff pressure.
5. The difference between pressures recorded in the two previous steps is then recorded as the measurement (in millimeters of mercury [mm Hg]) of pulsus paradoxus. A pulsus paradoxus >12 mm Hg is abnormal but nonspecific (see text).

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Cause of atrial fibrillation	
to remember it MITHA Mitral valvular heart disease Ischaemic heart disease Thyrotoxicosis H-hypertension A-Lone / idiopathic These are the important causes first told only this	<b>If you want what else then you tell the following</b> <ul style="list-style-type: none"> <li>• Alcohol</li> <li>• Cardiomyopathy</li> <li>• Congenital heart disease</li> <li>• Chest infection</li> <li>• Pulmonary embolism</li> <li>• Pericardial disease</li> <li>• Electrolyte imbalance</li> </ul>

Treatment of AF ?	
<b>If cardiac compromised if following are present</b> Rapid ventricular rate SBP < 90 mm of Hg Heart failure Impaired consciousness <b>TREATMENT is immediate cardioversion</b> <ul style="list-style-type: none"> <li>• <b>If not cardiac compromised</b></li> <li>• <b>Treatment is</b> <ul style="list-style-type: none"> <li>• Rate control</li> <li>• Rhythm control</li> <li>• Revert to sinus rhythm</li> </ul> </li> </ul>	<b>Drugs For AF</b> A. Amiodarone (rate control ) B. Beta-blocker (rate @ rhythm control) Metoprolol A. Calcium channel blocker (rate control) Verapamil or Diltiazem D--Digoxin (rate control—in structural heart Disease such as MS ) <b><u>If Thrombo-Embolism</u></b> Low molecular heparin (Inj. CardineX , Claxane ) 1 unit / kg bd for 5 days . Then Tab. Warin 5 mg or 2.5 mg 0 + 0 + 1 for 6 month ( maintain – INR 2-3)

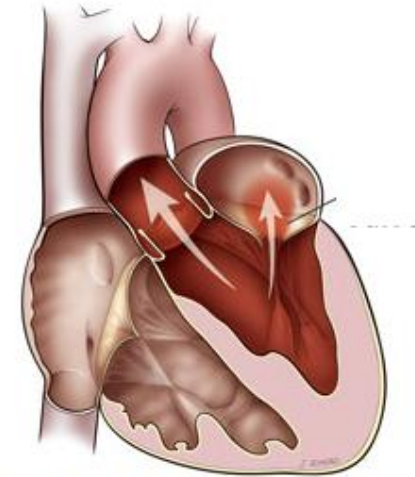
**HYPERTENSION**



**atrial**



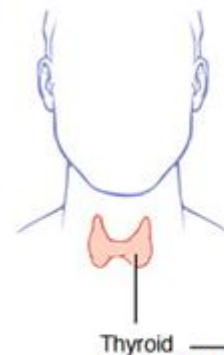
**MITRAL VALVE**



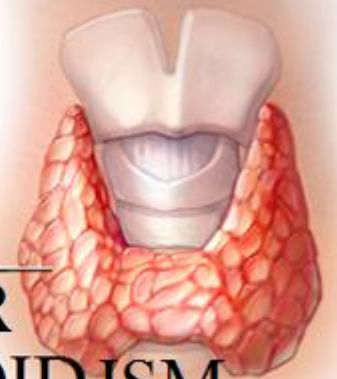
**fibrillation**

**IHD**

**Lone / idiopathic**



**HYPER  
THYROIDISM**



# QUESTION REGARDING APEX BEAT

1., what are the causes of impalpable apex beat?

To remember DOPER

- D—Dextrocardia
- O—Obesity
- P—Pericardial effusion
- E—Emphysema
- R—Behind the rib

Q. what are cause of shifting apex beat?

Normally apex remained 9 cm away from the mid line

Shift toward the left

**Due to heart cause :**

Cardiomegaly

- LVH---Apex shifted downward and laterally
- RVH---Apex shifted laterally

**Lung cause :**

Due to pushing :

- Right sided pleural effusion
- Right sided pneumothorax

Due pulling effect :

- Fibrosis and collapse of left lower Zone

Shifted toward the right :

**Lung cause :**

Due to pushing :

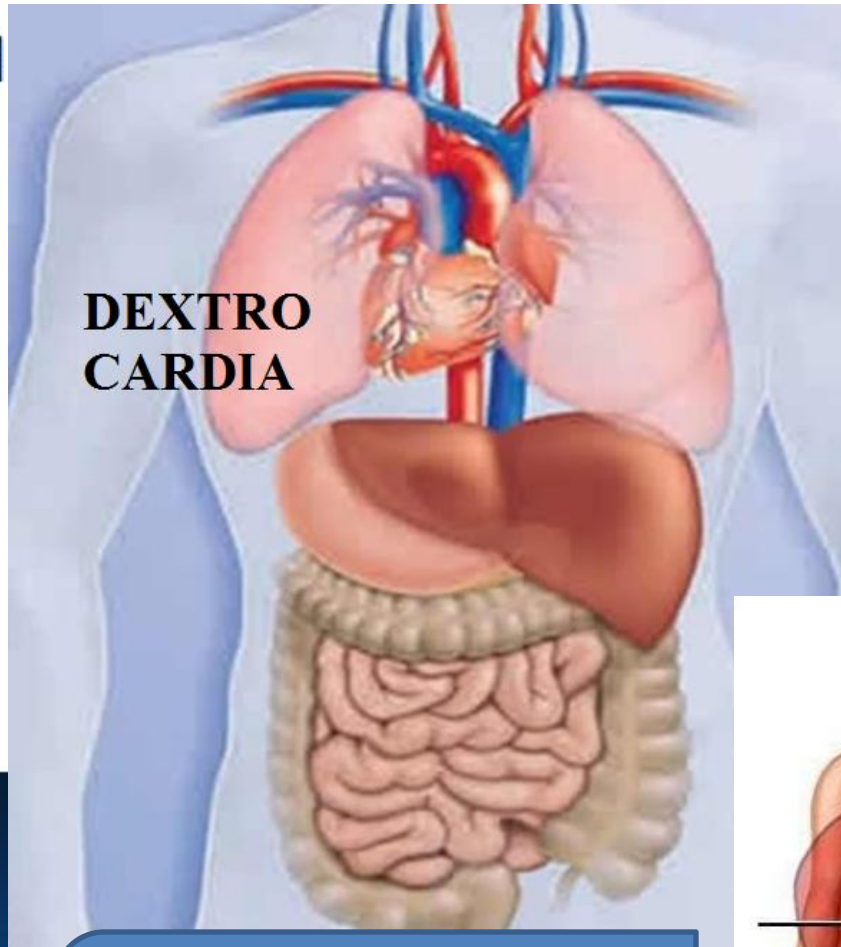
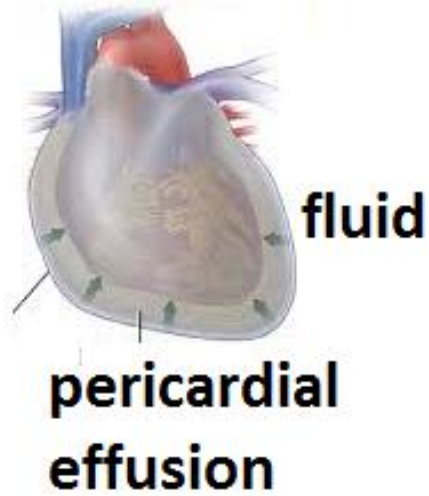
- Left sided pleural effusion
- Left sided pneumothorax

Due pulling effect :

- Fibrosis and collapse of right lower Zone



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Impalpable  
apex beat

DR SHAMOL



The apex beat is the most outer and downward cardiac impulse results from the left ventricle moving forward and striking the chest wall during systole.

What will u see during examination (palpation) of apex beat?

- Site ,Distance & character

How will you describe the apex beat after palpation ?

Apex beat is located in left 5<sup>th</sup> intercostal space 9 cm away from midline which is normal in character

**What r the character of apex beat?**

Character		Example
<b>Abnormal</b>		
Heaving	Forceful ,sustained and lift up finger	Due to pressure overload LVH due to <ul style="list-style-type: none"><li>• AS</li><li>• Systemic HTN</li></ul>
Thrusting	Forceful, less sustained and lift up finger	Due to volume overload Left ventricular dilatation <ul style="list-style-type: none"><li>• MR &amp;AR</li></ul>
Tapping	Neither forceful nor sustained and not lifted up finger	Mitral stenosis
Double apical impulse		Hypertrophy cardiomyopathy Ventricular aneurysm
Diffuse apex beat		MI Left ventricular aneurysm

QUESTION REGARDING LEFT PARA-STERNAL HEAVE  
&  
OTHER PULPATION

Name some condition where u may get left parasternal heave?	What is the feel of it?
Cause is right ventricular hypertrophy due to : <ul style="list-style-type: none"> <li>• Pulmonary HTN</li> <li>• Cor pulmonale</li> <li>• Pulmonary stenosis</li> <li>• Pulmonary regurgitation</li> <li>• Tricuspid regurgitation</li> </ul>	This is a sustained, thrusting pulsation usually felt at the left sternal edge indicating right ventricular enlargement
Name the condition where you may get palpable P2 ?	
Palpable P2 is found in pulmonary hypertension	

Name the condition where u may get epigastric pulsation ?	Name some condition where area of superficial cardiac dullness increased or decreased ?
<ul style="list-style-type: none"> <li>• Aneurysm of abdominal aorta</li> <li>• Pulsatile liver (TR)</li> <li>• Right ventricular hypertrophy –</li> <li>• Lean and thin person</li> <li>• Mass over lying abdominal aorta</li> </ul>	<b>Increased :</b> <ul style="list-style-type: none"> <li>• Pericardial effusion</li> <li>• Cardiomegaly (dilated cardiomyopathy )</li> </ul> <b>Decreased :</b> <ul style="list-style-type: none"> <li>• Emphysema</li> <li>• Pneumothorax</li> </ul>



# QUESTION REGARDING AUSCULTATION



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Q. write down the difference between first and 2<sup>nd</sup> heart sound

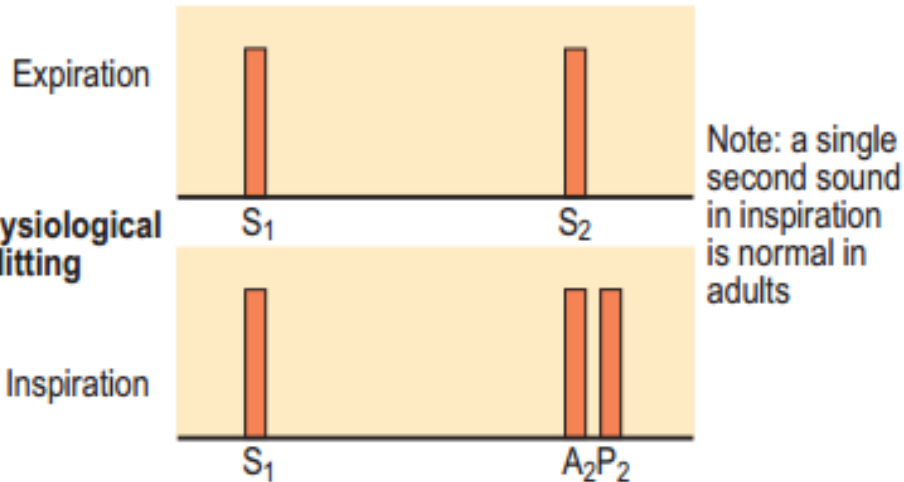
1 <sup>st</sup> heart sound	2 <sup>nd</sup> heart sound
• Due to closure of mitral and tricuspid valve	• Due to closure of aortic and pulmonary valve
• Low intensity	• High intensity
• More duration	• Less duration
• Coincide with carotid valve	• Just follow the carotid pulse
• Splitting absent	• Splitting present

loud first sound	loud 2 <sup>nd</sup> heart sound
Loud first heart sound <ul style="list-style-type: none"> <li>• Mitral stenosis</li> <li>• Tricuspid stenosis</li> <li>• Hyperdynamic circulation</li> </ul>	Loud 2 <sup>nd</sup> heart sound : <ul style="list-style-type: none"> <li>• Systemic HTN</li> <li>• Pulmonary HTN</li> </ul>
soft 1 <sup>st</sup> heart sound	soft 2 <sup>nd</sup> heart sound
Soft 1 <sup>st</sup> heart sound (RMC) <ul style="list-style-type: none"> <li>• R—Mitral regurgitation</li> <li>• M—Myocarditis</li> <li>• C ---Cardiomyopathy</li> </ul>	Soft 2 <sup>nd</sup> heart sound <ul style="list-style-type: none"> <li>• Calcified or severe aortic stenosis</li> <li>• Severe pulmonary stenosis</li> <li>• Aortic regurgitation</li> </ul>
Q what will be the intensity of 2 <sup>nd</sup> heart sound in aortic stenosis ? <ul style="list-style-type: none"> <li>• 2<sup>nd</sup> heart will be soft</li> </ul>	
Variable first heart sound	
Atrial fibrillation Extrasystoles Complete heart block	

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# Patho-physiology of splitting :

During inspiration	During expiration :
There is negative pressure in the thorax	Lung expand
↓--cause	↓--cause
Increased venous return	Pulmonary capillary also expand
↓ cause	↓--cause
Increased right ventricular end diastolic volume	Increased capacity of pulmonary circulation
↓cause	↓--cause
So delayed closure of pulmonary valve	Blood pooling into pulmonary Vessel
You will get splitting	↓--cause
	Decrease venous return into left atrium
	↓--cause
	Decrease left ventricular end diastolic volume
	↓--cause
	Early closure of aortic valve





Q cause of wide splitting

Widens in inspiration (enhanced physiological splitting :

- Right bundle branch
- Pulmonary stenosis
- Pulmonary hypertension
- ventricular septal defects

Fixed  
splitting of  
second  
sound

Expiration



Inspiration



True fixed splitting is a characteristic of atrial septal defect. Increased splitting occurs in right bundle branch block and pulmonary hypertension. It is not fixed.

Q. wide and fixed splitting

Cause of wide and fixed splitting

- Atrial septal defect

Q cause of reverse splitting

Cause of reverse splitting

- Aortic stenosis
- Hypertrophic cardiomyopathy
- Left bundle branch block
- ventricular pacemaker

Reversed  
splitting of  
second  
sound

Expiration



Inspiration



Reversed splitting occurs in left ventricular outflow obstruction and left bundle branch block

Causes of a third heart sound	Cause of 4 <sup>th</sup> heart sound
Physiological <ul style="list-style-type: none"> <li>• Athletes</li> <li>• Pregnancy</li> <li>• Fever</li> </ul>	A fourth heart sound may be heard in <ul style="list-style-type: none"> <li>• Left ventricular hypertrophy,</li> <li>• Hypertension and</li> <li>• Aortic stenosis.</li> </ul>
Pathological <ul style="list-style-type: none"> <li>• LVF</li> </ul>	
<ul style="list-style-type: none"> <li>• Cause of third heart sound – Rush of blood from Atria to Ventricle during rapid filling phase of Cardiac Cycle.</li> </ul>	Due to Atrial contraction which causes rapid flow of blood from Atria to noncompliant Ventricle and vibration in the blood
It is soft and low-pitched, best heard with the stethoscope bell at the apex. It occurs just after S1 (lub-da-dub;)	It is soft and low-pitched, best heard with the stethoscope bell at the apex. It occurs just before S1 (da-lub-dub).
<ul style="list-style-type: none"> <li>• What is gallop rhythm</li> <li>• In heart failure S3 occurs with a tachycardia, referred to as a ‘gallop’ rhythm, and S1 and S2 are quiet (lub-da-dub;)</li> </ul>	

## Comparing the 3rd and 4th heart sounds

LearnTheHeart.com

<b>S3 - "ventricular gallop"</b>	<b>S4 - "atrial gallop"</b>
Occurs in early diastole Occurs during passive LV filling May be normal at times Requires a very compliant LV Can be a sign of systolic CHF	Occurs in late diastole Occurs during active LV filling Almost always abnormal Requires a non-compliant LV Can be a sign of diastolic CHF



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What is murmur?

Murmur is the abnormal sound produce by either normal amount blood passing through abnormal valve or increased or abnormal amount of blood passing through the normal valve

Classify the murmur?

Systolic murmur	Diastolic	Continuous
1. Ejection systolic 2. Pansystolic 3. Late systolic	1. Early diastolic 2. Mid diastolic	1. PDA—patent ductus arteriosus

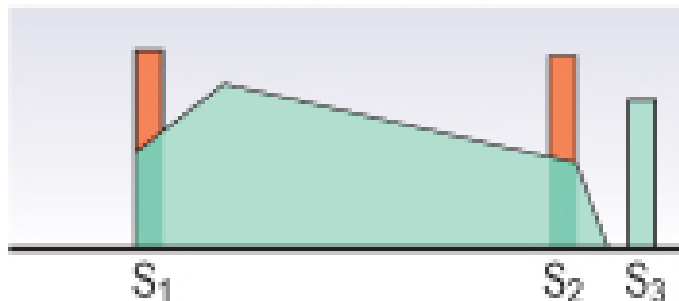
Write down the feature of benign or innocent murmur?

- Soft
- Mid systolic
- Heard at left sternal edge
- Nor radiation

Give some example of systolic murmur

Pan systolic murmur

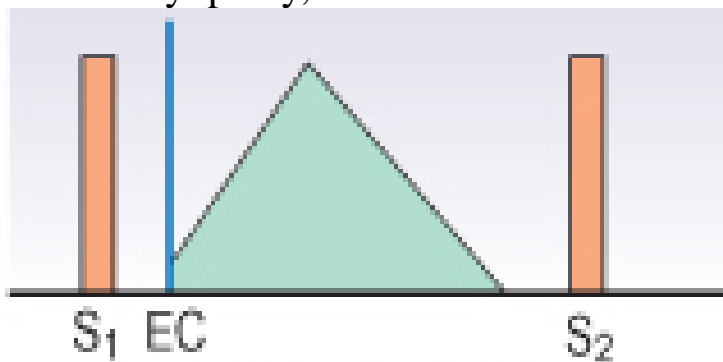
1. Mitral regurgitation
2. Tricuspid regurgitation
3. Ventricular septal defect
4. aortopulmonary shunts



mechanism = Flow between two chambers that have widely different pressure throughout systole

Ejection systolic

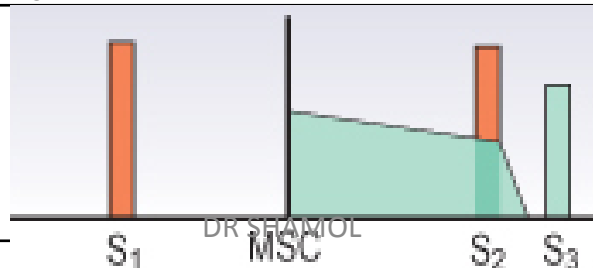
- Aortic stenosis
- Pulmonary stenosis
- Aortic sclerosis,
- hypertrophic obstructive cardiomyopathy,
- atrial septal defect,
- flow murmurs
  - fever,
  - pregnancy,
  - hyperthyroidism,
  - anemia,
  - aortic regurgitation due to high flow



mechanism Crescendo—decrescendo in shape (the sound of a 'woodcutter sawing wood in a forest') as pressure rises and falls

Late systolic

- Left atrial myoxma



mechamism = Mitral regurgitation after the valve has clicked open in mitral valve prolapse

Give some example of diastolic murmur

### Early diastolic

Aortic regurgitation

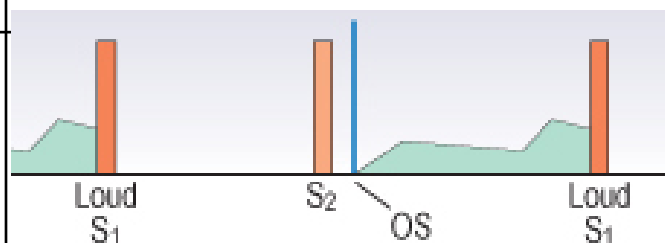
Pulmonary regurgitation



mechanism = Begins with or shortly after S2 as soon as ventricular pressure falls below pressure in the aorta or pulmonary artery .High-pitched, whispering and decrescendo

### Mid diastolic

1. Mitral stenosis
2. Carey comb murmur (Mitral valvulitis in rheumatic fever)
3. Tricuspid stenosis
4. ASD
5. Austin flow murmur



Disproportion between valve orifice and flow rate Carey Coombs murmur of acute rheumatic fever due to mitral valve inflammation In severe aortic regurgitation, a mid-diastolic murmur (Austin Flint murmur) may arise at then anterior mitral valve leaflet as jets of blood from the aortic root and left atrium collide

### CONTINUOUS MURMURS—

1. patent ductus arteriosus,
2. arteriovenous fistula,
3. aortopulmonary connection,
4. venous hum

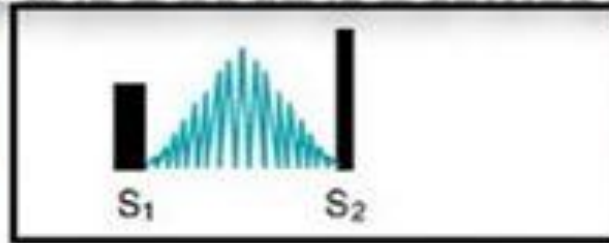
causes of presystolic accentuation of murmurs Typical of mitral stenosis when the pressure-overloaded left atrium forcefully contracts and occurs only in sinus rhythm

## MURMURS

@doctordconline

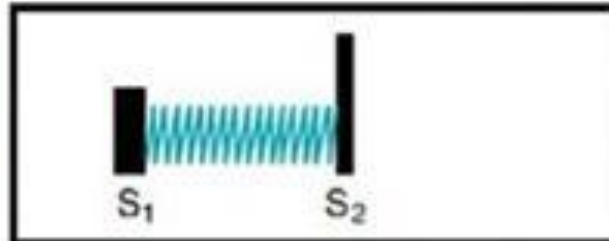
## DISEASES

### EJECTION SYSTOLIC MURMUR



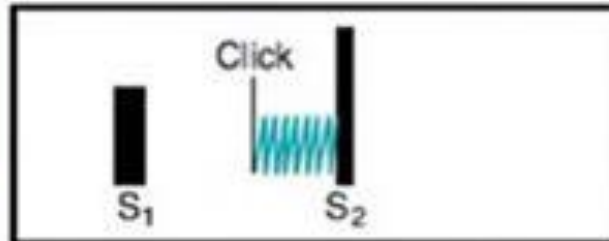
- Aortic stenosis
- Pulmonary stenosis

### PANSYSTOLIC MURMUR



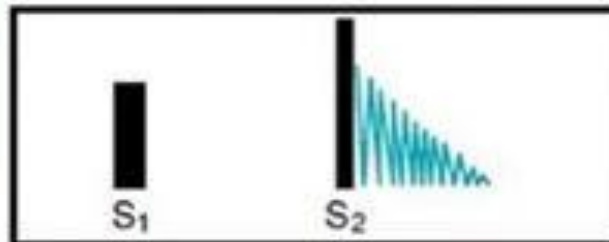
- Mitral regurgitation
- Tricuspid regurgitation
- Ventricular septal defect

### MIDSYSTOLIC MURMUR



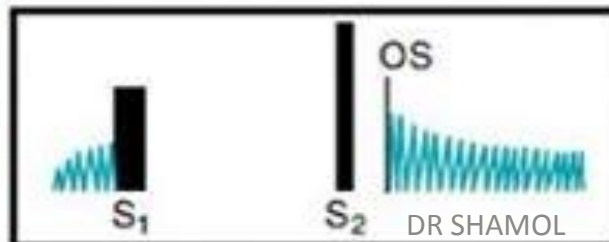
- Mitral valve prolapse

### EARLY DIASTOLIC MURMUR



- Aortic regurgitation

### MID-DIASTOLIC MURMUR



- Mitral stenosis
- Tricuspid stenosis

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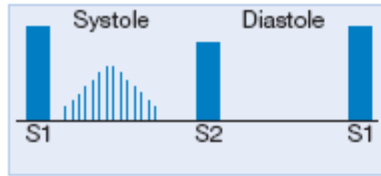
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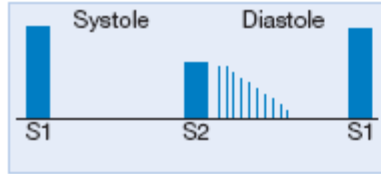
## AORTIC VALVE

③

Stenosis



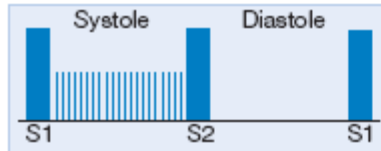
Regurgitation



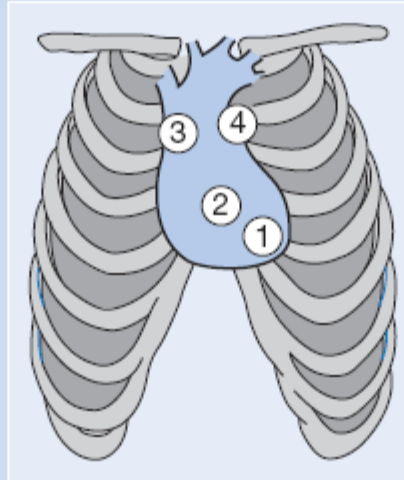
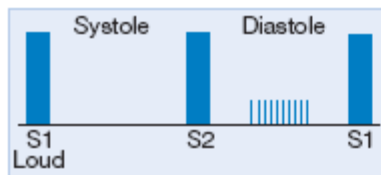
## TRICUSPID VALVE

②

Regurgitation



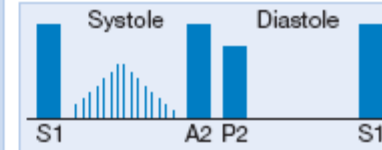
Stenosis



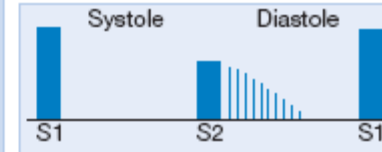
## PULMONARY VALVE

④

Stenosis



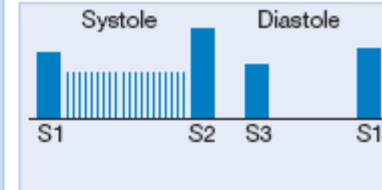
Regurgitation



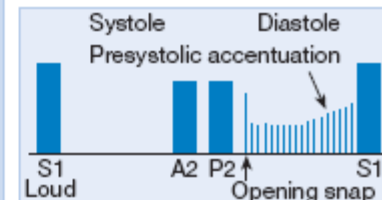
## MITRAL VALVE

①

Regurgitation



Stenosis



	MS	MR	AS	AR
Pulse			Low volume Slow rising	High volume Collapsing
Apex beat	Tapping & not shifted	Thrusting & shifted	Heaving & not shifted	Thrusting & shifted
Heart sound 1 <sup>st</sup>	Loud	Soft	N	N
Heart sound 2 <sup>nd</sup>	N	N	SOFT	SOFT
Murmur	MDM	Pan systolic	Ejection systolic	Early diastolic
Area	Mitral	Mitral	Aortic	Tricuspid or left lower sternal
Radiation	X	Axilla	Neck	X

## What will u see during describe a murmur

### 1. Character (**Ch**)

- rough , rumbling,
- MR--- Loud ,blowing
- AR----- High pitch blowingh
  - ---Harsh , high pitched and musical
  - VSD
  - ASD
  - PAD-- Loud, continuous 'machinery' murmur , train in tunnel

### 1. Intensity or loudness—see the grading (**I**)

### 2. Timing --Systolic / diastolic (**T**)

- 

### 3. Radiation (**Ra** )

- MR----toward the axilla
- AS ----towarr to right neck

### 4. Relation with respiration (**Ranjan**)

- Right sided murmur increased in inspiration (**PS , TR** )

### 5. left sided murmur increased in expiration Site or location--- (**Sir**)

- MS & MR---Mitral area
- AR-----tricuspid area
- AS----aortic area
- PS ----pulmonary area
- TR & VSD---tricuspid area & left lower parasternal area (3<sup>rd</sup> & 4<sup>th</sup> space )
- PAD

- To remember it **Chitra ranjan sir**

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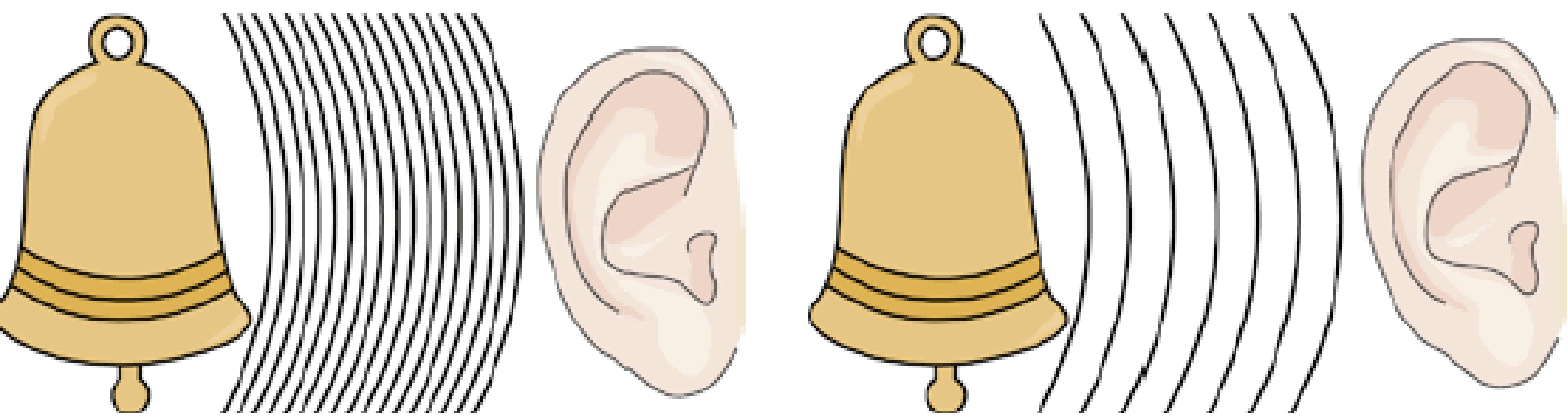
Right sided murmur (PS , TR )	increased in inspiration & or sustained abdominal pressure (↑venous return)
left-sided murmurs (AS,AR) (MS,MR)	louder during expiration
Valsalva maneuver	↓venous return and ↑systemic arterial resistance
	most murmurs decrease in length and intensity during the Valsalva
	But systolic murmur of HCM & mitral valve prolapse becomes much louder
standing	reduced preload
	most murmurs diminish in intensity
	but of HCM become louder and
	murmur of mitral valve prolapse lengthens and intensified
Squatting or passive leg raising	both ↑ venous return and ↑ systemic arterial resistance
	produces opposite effect of standing
isometric exercise (sustain handgrip exercise for 20–30 seconds))	↑systemic arterial resistance & heart rate
	murmurs caused by blood flow across normal or obstructed valves (e.g. mitral or pulmonic stenosis) become louder
	Murmurs of mitral and aortic regurgitation and ventricular septal defect also increase
	murmur of AS,HCM,mitral prolapsed softer





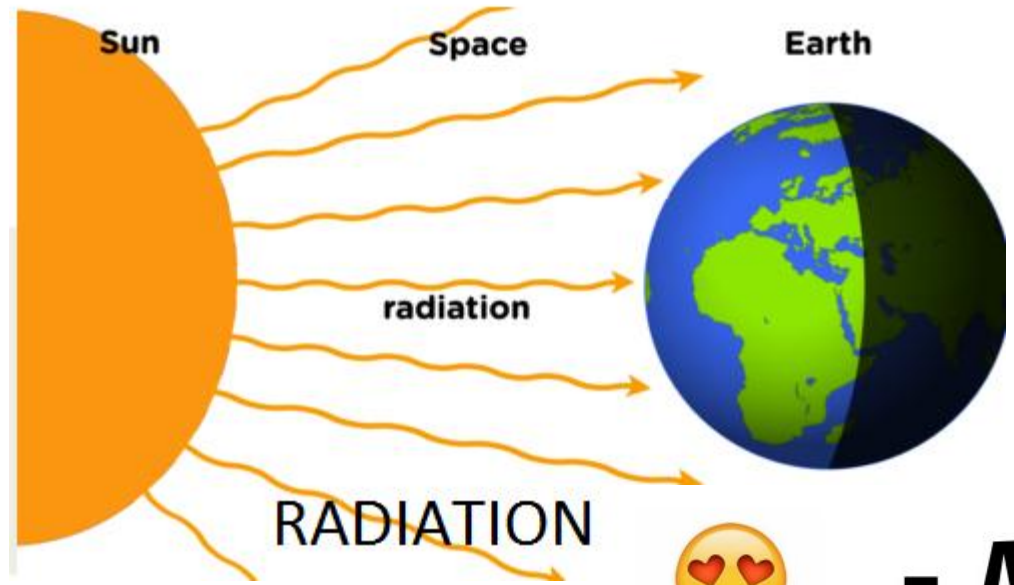
**TIMING**

**DR. SHAMOL**



**INTENSITY OR LOUDNESS**

DR. SHAMOL



= A



= B



= C

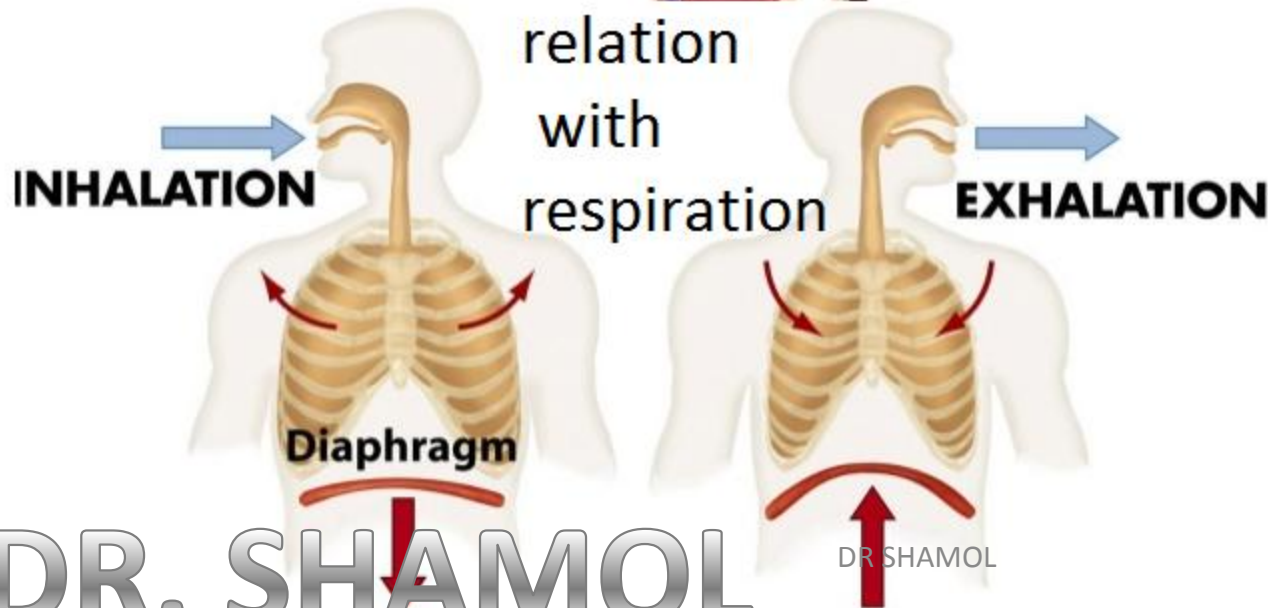


= D



= F

GRADING



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CHARACTER

Grading of murmur		
Grade		Thrill
1	Heard by an expert in optimum conditions	no thrill
2	Heard by a non-expert in optimum conditions	no thrill
3	Easily heard	no thrill
4	A loud murmur	with a thrill
5	Very loud, often heard over wide area	with thrill
6	Extremely loud, heard without stethoscope	

Only I can hear

G1

Heard by an expert in optimum conditions



Sir we can hear

G2

Heard by a non-expert in optimum conditions



mid level in cabin

professor in cabin

G3

Easily heard

He! He! we can Also hear



inter doctor

G4

LOUD



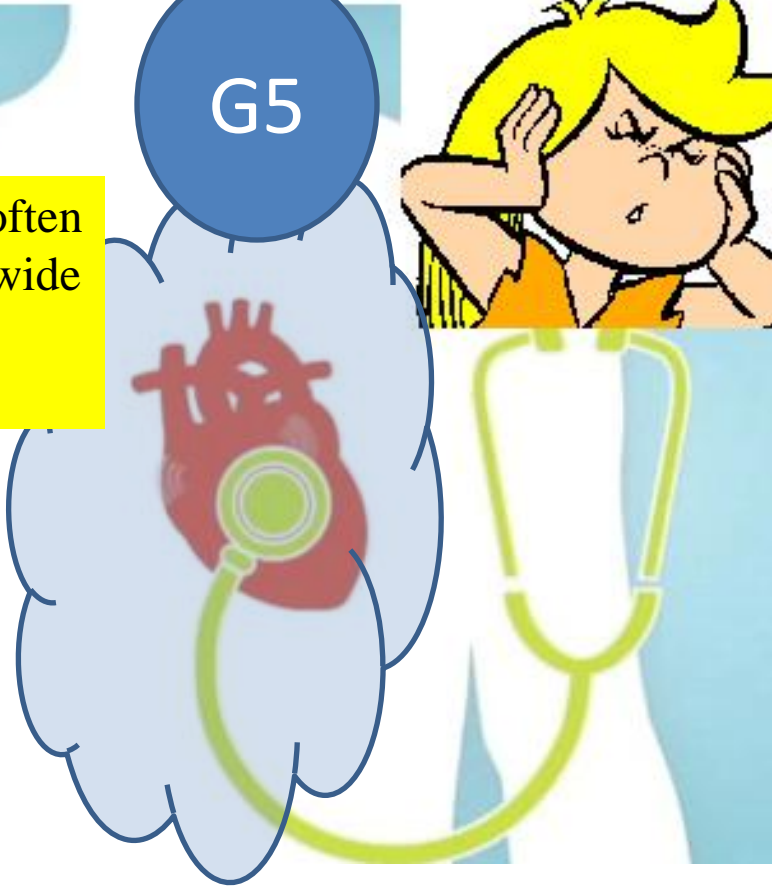
A loud murmur

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G5

Very loud, often  
heard over wide  
area



i can hear

G6



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MS	<ul style="list-style-type: none"> <li>• Low pitched , localized , rough , rumbling,</li> <li>• Mid diastolic murmur ,</li> <li>• Best heard with bell of stethoscope in left lateral position and breath hold expiration</li> </ul>
MR	<ul style="list-style-type: none"> <li>• Loud ,blowing in character ,</li> <li>• Pansystolic murmur best heard at the apex radiate to axilla</li> </ul>
AS	<p>Harsh , high pitched and musical</p> <p>Ejection systolic murmur</p> <p>usually audible all over the precordium but more prominent is aortic area and radiate to right side of the neck .The murmur is often likened to a saw cutting wood and may be (especially in older patients) have a musical quality like the ‘mew’ of a seagull</p>
AR	<ul style="list-style-type: none"> <li>• High pitch blowingh early diastolic murmur</li> <li>• Best heard left lower para stenal area (3<sup>rd</sup> or 4<sup>th</sup> space )</li> <li>• with patient sitting and leaning forward and breath hold after expiration</li> </ul>
VSD	pansystolic murmur, usually heard best at the left sternal edge but radiating all over the precordium
ASD	Ejection systolic flow murmur in the left second and third intercostals space (due to increase flow to pulmonary valve )
PDA	<ul style="list-style-type: none"> <li>• Loud, continuous 'machinery' murmur , train in tunnel</li> <li>• Pansystolic murmur</li> <li>• heard along the left upper sternal border and outer border of the clavicle.</li> </ul>

Added sounds	
Opening snap (OS)	This is a brief, high-pitched, early diastolic sound occurring after S2 as the mitral valve is forced open by high left atrial pressure in mitral stenosis. The S2–OS interval is shorter the higher the left atrial pressure.
Ejection click or sound	This is a sharp, high-pitched sound in early systole soon after S1. It may occur in non-calcified aortic stenosis or pulmonary stenosis with a pliable valve, the mechanism similar to an opening snap and the sound preceding the typical ejection systolic murmur.
Mid-systolic (non-ejection) click	This may occur in mitral valve prolapse as one or both leaflets prolapse into the left atrium. There may be an accompanying late systolic murmur of mitral regurgitation.
Metallic prosthetic sounds	These may be audible without auscultation.
Pericardial knock	This is a third-heart-sound equivalent heard in diastole in constrictive pericarditis
Pericardial friction rub	This is a scratching presystolic, systolic or early diastolic sound heard best with the diaphragm. It is due to pericarditis

**TABLE 7-1****Effects of Maneuvers on Valvular Murmurs**

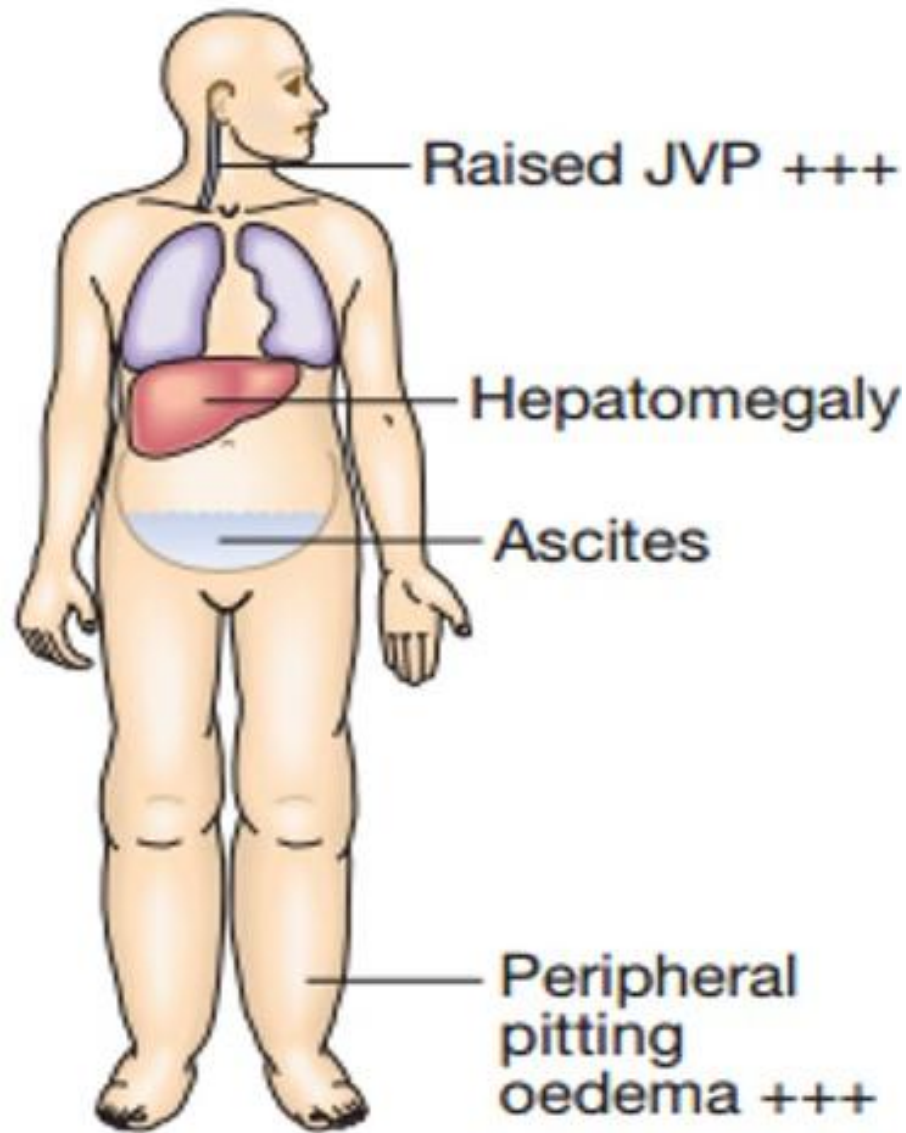
MANEUVERS: EFFECT ON MURMUR INTENSITY*				
Valve Abnormality	Valsalva (during Continuous Strain)	Amyl Nitrite	Handgrip	Squatting
Aortic stenosis	↓	↑	↓	↑
Hypertrophic cardiomyopathy (see Chapter 6)	↑	↑	↓	↓
Chronic aortic regurgitation	↓	↓	↑	↑
Chronic mitral regurgitation	↓	↓	↑	↑
Mitral valve prolapse	Moves click and murmur onset closer to S <sub>1</sub>	Moves click and murmur onset closer to S <sub>1</sub>	Moves click and murmur onset closer to S <sub>2</sub>	Moves click and murmur onset closer to S <sub>2</sub>
Mitral stenosis	↓	↑	↑	↑

\*Valsalva maneuver: During continuous strain, increases intrathoracic pressure, thereby decreasing venous return and preload. After strain, arterial

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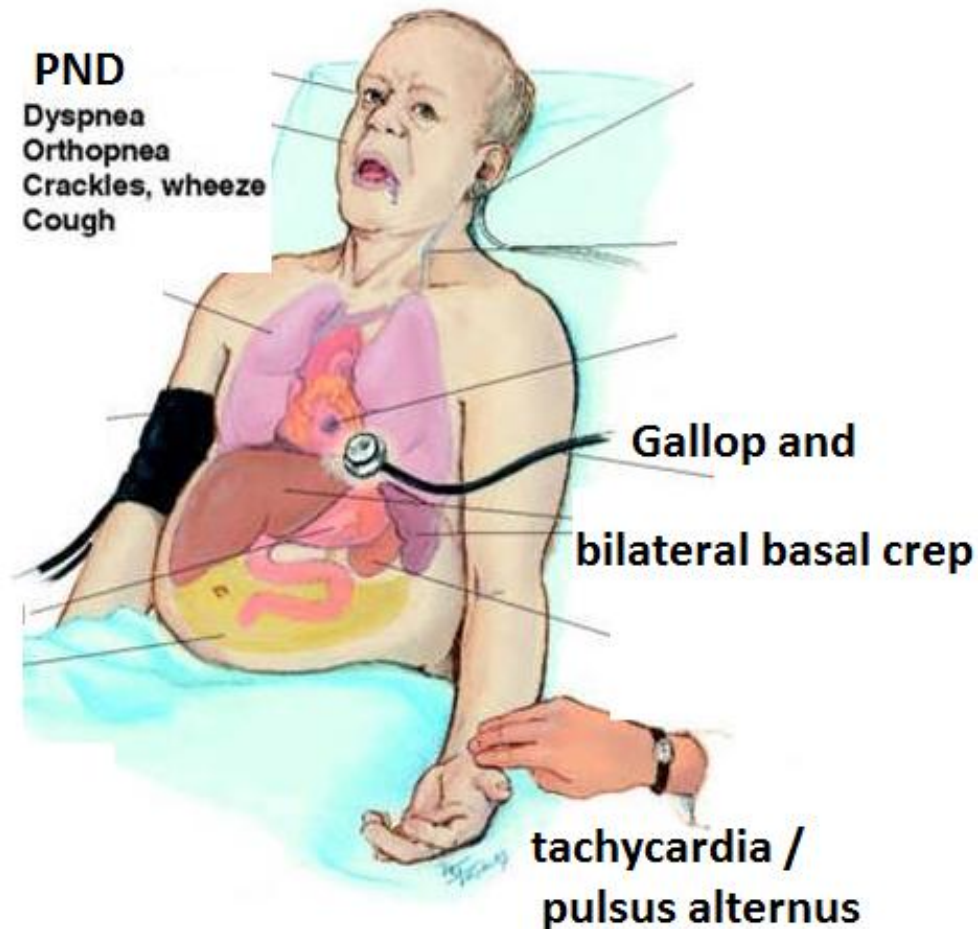


**Right**

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**PND**

Dyspnea  
Orthopnea  
Crackles, wheeze  
Cough



Left heart failure

# LEFT SIDED ♥ FAILURE

- 
- Paroxysmal Nocturnal Dyspnea
  - Elevated Pulmonary Capillary Wedge Pressure
  - Pulmonary Congestion
    - Cough
    - Crackles
    - Wheezes
    - Blood-Tinged Sputum
    - Tachypnea
  - Restlessness
  - Confusion
  - Orthopnea
  - Tachycardia
  - Exertional Dyspnea
  - Fatigue
  - Cyanosis

# RIGHT SIDED ♥ FAILURE

(Cor Pulmonale)



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	Pressure	Valve stenosis	VOLUME OR regurgitated	Muscle
Right heart Failure	Pulmonary hypertension	PS	TR,	Cardiomyopathy , IHD, MI Myocarditis
	Cor-pulmonale (COPD)		Volume overload , IV fluid , anaemia , thyrotoxicosis	
Left heart failure	Systemic hypertension	AS	AR , MR	

Cardinal feature of right heart failure	Cardinal feature of left ventricular failure
<ul style="list-style-type: none"> <li>Depended edema</li> <li>Tender hepatomegaly</li> <li>Raised JVP</li> </ul>	<ul style="list-style-type: none"> <li>Tachycardia or pulsus alternans</li> <li>Cyanosis</li> <li>Gallop rhythm</li> <li>Bilateral basal creps (+)</li> </ul>



<b>Treatment of RVF</b> <ul style="list-style-type: none"> <li>• Bed rest</li> <li>• Low flow Oxygen inhalation</li> <li>• Diuretic</li> <li>• Treatment of underlying causes</li> </ul>	<b>Treatment of LVF</b> <ul style="list-style-type: none"> <li>○ Bed rest in propped up position</li> <li>○ High flow Oxygen inhalation</li> <li>○ Inj . Lasix 2 amp IV stat and up 8 amp or 160 mg</li> <li>○ Treatment of the cause</li> </ul>
--	--

	New York Heart Association (NYHA) functional classification—no sit MU	
Class I	no—no limitation	No limitation during ordinary activity
Class II	sit –slight limitation	Slight limitation during ordinary activity
Class III	m-- Marked limitation	Marked limitation of normal activities without symptoms at rest
Class IV	u—Unable /rest symptom	Unable to undertake physical activity without symptoms; symptoms may be present at rest

# NYHA CLASS



## Class I

No symptoms  
Can perform ordinary activities without any limitations

## Class II

Mild symptoms  
Occasional swelling  
Somewhat limited in ability to exercise or do other strenuous activities  
No symptoms at rest

## Class III

Noticeable limitations in ability to exercise or participate in mildly strenuous activities  
Comfortable only at rest

## Class IV

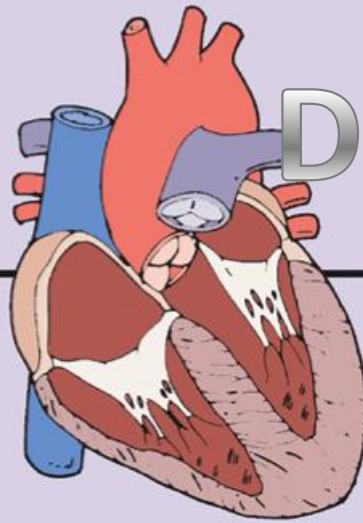
Unable to do any physical activity without discomfort  
Symptoms at rest



cor pulmonale	is “hypertrophy of the right ventricle(with or without failure )resulting from diseases affecting the function and/or structure of the lung, except when these pulmonary alterations are the result of diseases that primarily affect the left side of the heart or congenital heart disease.”
---------------	--

### What are the causes of cor-pulmonale ?

Diseases of the Lung Parenchyma	<ul style="list-style-type: none"> <li>➤ Chronic obstructive pulmonary disease <ul style="list-style-type: none"> <li>○ Emphysema</li> <li>○ Chronic bronchitis</li> </ul> </li> <li>➤ Cystic fibrosis</li> <li>➤ ILD</li> <li>➤ Sarcoidosis</li> <li>➤ Bronchiectasis</li> </ul>
Disorders of Chronic (Alveolar) Hypoxia	<ul style="list-style-type: none"> <li>➤ Neuromuscular respiratory failure</li> <li>➤ Chest wall disorders <ul style="list-style-type: none"> <li>○ Kyphoscoliosis, AKS</li> </ul> </li> <li>➤ Alveolar hypoventilation syndromes</li> <li>➤ Sleep apnea syndrome (e.g. Pickwickian syndrome of extreme obesity with hypoventilation).</li> </ul>
Diseases of the Pulmonary Vasculature	<p>Pulmonary arterial hypertension (PAH)</p> <p>Venoocclusive disease</p> <p>Chronic thromboembolic pulmonary hypertension</p>



## Right heart failure

### Congestion of peripheral tissues

Dependent edema and ascites

### Liver congestion

Signs related to impaired liver function

### GI tract congestion

Anorexia, GI distress, weight loss

## Left heart failure

### Decreased cardiac output

Activity intolerance and signs of decreased tissue perfusion

### Pulmonary congestion

### Impaired gas exchange

Cyanosis and signs of hypoxia

### Pulmonary edema

Orthopnea

Cough with frothy sputum

Paroxysmal nocturnal dyspnea



## **In short case u have to face following?**

Auscultation of a patient with murmur (MS or MR or Mixed)—  
very rare only give if the case is available

In normal patient u have examination following

- Inspect the precordium

- Palpate the precordium or locate the apex beat

- Show me auscultation or show me the murmur

- show me the thrill

- as a whole examine the precordium

in some case u may to ask examine the peripheral pulse

JVP

blood pressure

You have to read –MS, MR, AS, AR, TOF and

do not go to exam hall with out reading MS and AF

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# Short case MS

Normal  
mitral valve

Narrow  
mitral valve

Left atrium

Left ventricle

M

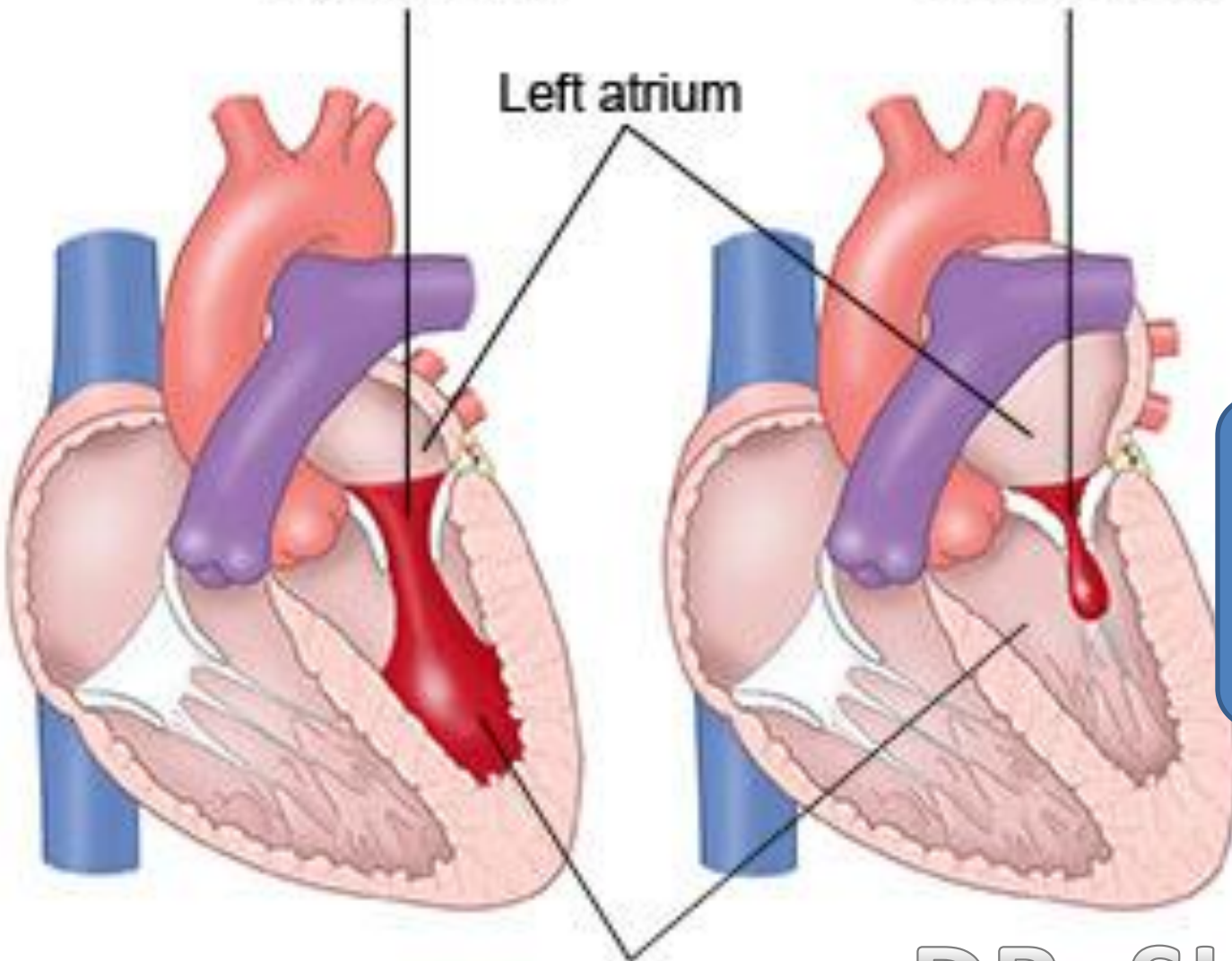
S

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Normal Heart

Mitral Valve Stenosis

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## What is MS?

Mitral stenosis (MS), an obstruction to blood flow between the left atrium (LA) and the left ventricle (LV), is caused by abnormal mitral valve function

## What is the causes of mitral stenosis ?

chronic rheumatic heart disease –commonest

congenital

calcified

carcinoid syndrome

Connective tissue disease (most commonly SLE,RA)

Drugs: methysergide

Mucopolysaccharidoses

## Some percentage regarding MS?

How many patients with rheumatic mitral valve disease do not give a history of rheumatic fever or chorea

60%

How many patients of patients with acute rheumatic carditis do not develop clinical valvular heart disease

about 50%.

How many patients with rheumatic heart disease have isolated MS

40%



symptoms	mnemonic		reason behind it
	<b>A</b>	angina /Chest pain	pulmonary hypertension
	<b>B</b>	Breathlessness	pulmonary congestion
	<b>C</b>	Cough	pulmonary congestion
	<b>D</b>	disease causes by MS or its complication	Thromboembolic complications (e.g. stroke, ischaemic limb)
	<b>E</b>	edema, ascites	right heart failure
	<b>F</b>	Fatigue	low cardiac output
	<b>H</b>	Haemoptysis	pulmonary congestion, pulmonary embolism
		hoarseness appears (Ortner syndrome)	if LA becomes so large that it impinges on the left recurrent laryngeal nerve,
	<b>GP</b>	Palpitation	atrial fibrillation

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## Examination finding of MS for MBBS

Pulse	Irregular / pulsus deficit –due AF
-------	------------------------------------

Bp	Normal
----	--------

### Examination of precordium

Inspection	Normal or visible apex beat
------------	-----------------------------

Palpation	<ul style="list-style-type: none"><li>• Apex –not shifted and tapping in nature</li><li>• Left parasternal heave –if RVH</li><li>• Palpable P<sub>2</sub> ----in pulmonary HTN</li></ul>
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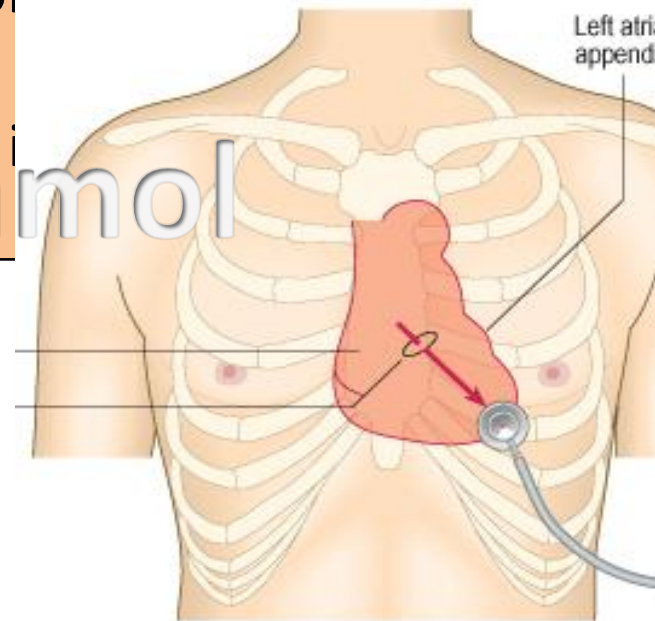
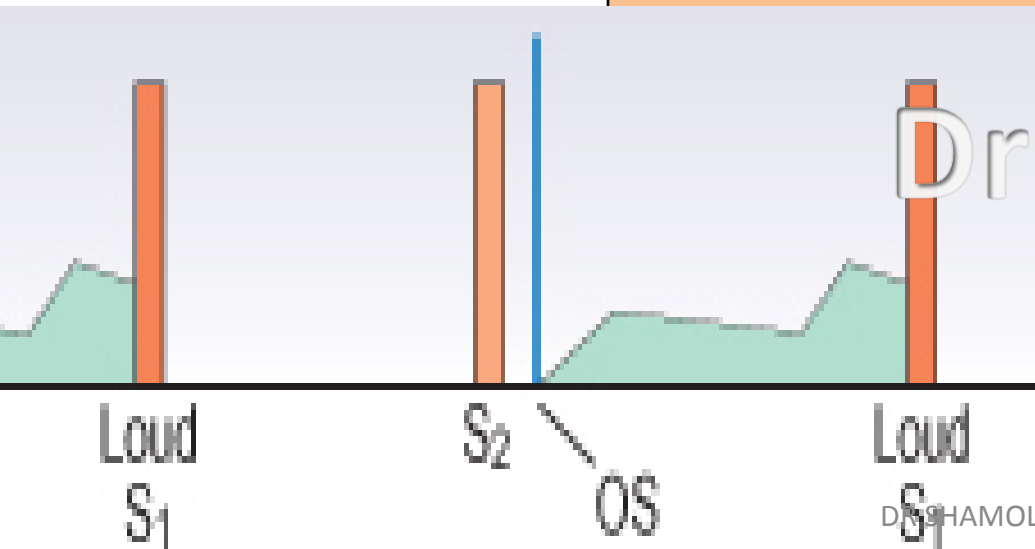
Auscultation	<ul style="list-style-type: none"><li>• Loud first heart sound (in case of AF—heart sound of variable intensity )</li><li>• Opening snap</li><li>• Low pitched , localized , rough , rumbling Mid diastolic murmur , with pre systolic accentuation</li><li>• Best heard with bell of stethoscope in left lateral position and breath hold expiration</li></ul>
--------------	---

Three think never forget

Tapping apex beat that not shifted  
( if shifted then it associated with MR)

Loud first heart sound ( Tell it variable intensity if AF)

- ❖ characteristic MID DIASTOLIC MURMUR
- ❖ Low pitched , localized , rough , rumbling Mid diastolic murmur , with pre systolic accentuation
- ❖ Best heard with bell of stethoscope in left lateral position and breath hold expiration
- ❖ Presystolic accentuation -- absent if associated



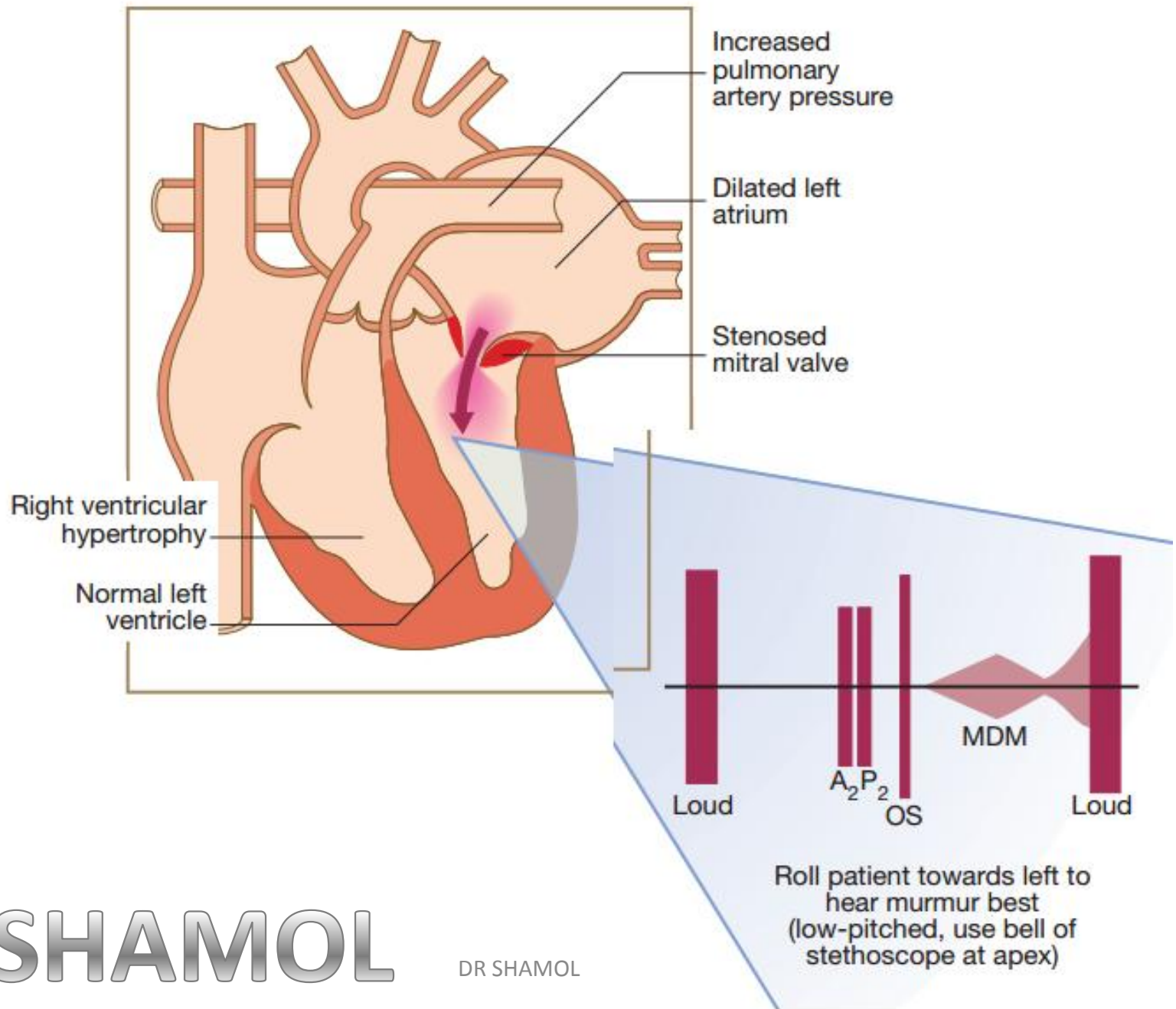
## For postgraduate

sign	general exam	Mitral facies/Malar flash			
		<b>pulse</b>	is regular ,low volume		
			irregularly irregular (from atrial fibrillation)		
		<b>JVP</b>	raised if Right heart failure		
		<b>edema</b>	if right heart failure		
	<b>precordium</b>	<b>palpation</b>	apex beat	Tapping in nature and situated in the 5th intercostal space just medial to midclavicular line	
			palpable P2	if pulmonary hypertension	
			Left parasternal heave	indicating right ventricular enlargement	
			Diastolic thrill	at apex.	
		<b>Auscultation</b>	heart sound	Loud first heart sound	
				loud P2—if pulmonary hypertension	
				If AF—1 <sup>st</sup> sound will be variable intensity	
			added sound	Low pitched, rough rumbling, mid-diastolic murmur with pre-systolic accentuation (if in sinus rhythm) is heard best at the apex with the patient in a left lateral position	
				Early diastolic murmur due to pulmonary regurgitation from pulmonary hypertension (Graham Steell murmur)	
				opening snap	
				Creptitations--	if pulmonary oedema
				Effusions (raised pulmonary capillary pressure)	

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## What is the characteristic of murmur?

Rumbling, low-pitched, mid-diastolic murmur, best heard in the left lateral position on expiration. In sinus rhythm there may be presystolic accentuation of the murmur. presystolic accentuation is absent in atrial fibrillation

If you are not sure about the murmur, tell the examiner that you want the patient to perform sit-ups or hopping on one foot to increase the heart rate. This will increase the flow across the mitral valve and the murmur is better heard

sequence of auscultatory finding?

loud S1 → P2 accentuated (PHTN) → Opening snap (OS) → Low pitched, rough rumbling, mid-diastolic murmur with → pre-systolic accentuation → best at the apex with the patient in a left lateral position after expiration

if AF → 1<sup>st</sup> heart sound become variable intensity and pre-systolic accentuation absent

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## **What are the complication of MS?**

Firs utter the following 4 cause then other s (CASE)-history

<b>(A)</b>	Atrial fibrillation
<b>(S)</b>	Stroke /Systemic Thrombo-embolism
<b>(C)</b>	Right heart failure or CCF (due to pulmonary hypertension )
<b>(E)</b>	pulmonary edema
<b>H/ History</b>	pulmonary hypertension

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Picture

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complication	<b>A</b>	Atrial fibrillation.	
	<b>B</b>	Ball valve thrombus (which is a big thrombus) may lead to sudden death.	
		Bronchitis and recurrent bronchopulmonary infection	
	<b>C</b>	Chest pain in 10% cases (due to pulmonary hypertension).	
	<b>D</b>	Dysphagia due to enlarged left atrium	
	<b>E</b>	Pulmonary edema (left atrial failure).	
	<b>F</b>	Failure –CCF –due to PH	
	<b>H</b>	Hemoptysis.	
	<b>I</b>	ILD --Long standing MS may cause interstitial lung disease (due to prolonged pulmonary edema) and hemosiderosis	
	<b>T</b>	Thrombo-embolism	systemic embolism—commonly cerebral (cerebral infarction with hemiplegia)
			also in mesenteric ischaemia , renal
			peripheral./limb ischaemia
	<b>O</b>	Ortner's syndrome	enlarged left atrium gives pressure on left recurrent laryngeal nerve, causing hoarseness of voice.
	<b>P</b>	Pulmonary hypertension	
		Pulmonary congestion, embolism, infarction	

<b>What is tapping apex beat?</b>	
It is the accentuated, palpable 1st heart sound	
<b>Why first sound is loud?</b>	
closure of the mitral valve against increase left atrial pressure	
<b>when loudness disappear ?</b>	
when the valve become heavily calcified	
<b>What is opening snap?</b>	It is a short, sharp high pitched sound, heard immediately after 2nd heart sound (during diastole), produced by sudden opening of mitral valve, due to raised left atrial pressure
<b>where found</b>	Opening snap is usually found in MS.
<b>absent when</b>	when valve become calcified
<b>significant</b>	It indicates that the valve cusp is still mobile
	It is absent when the valve is calcified
	The gap between second heart sound and opening snap indicates the severity of MS.
	The diminishing gap indicates severe MS
	It is always due to organic MS
<b>pre-systolic accentuation</b>	It is due to atrial systole, which increases the flow across the stenosed mitral valve from left atrium to left ventricle, causing accentuation or loudness of the murmur
<b>when present</b>	Is present only if there is sinus rhythm..
<b>when absent</b>	It is absent, if there is atrial fibrillation
<b>in MS apex beat is shifted or not ?</b>	usually not shifted as in MS left ventricle not enlarge only left atrium is enlarge
<b>in MS if u got apex beat shifted then what will you think ?</b>	The patient has associated MR

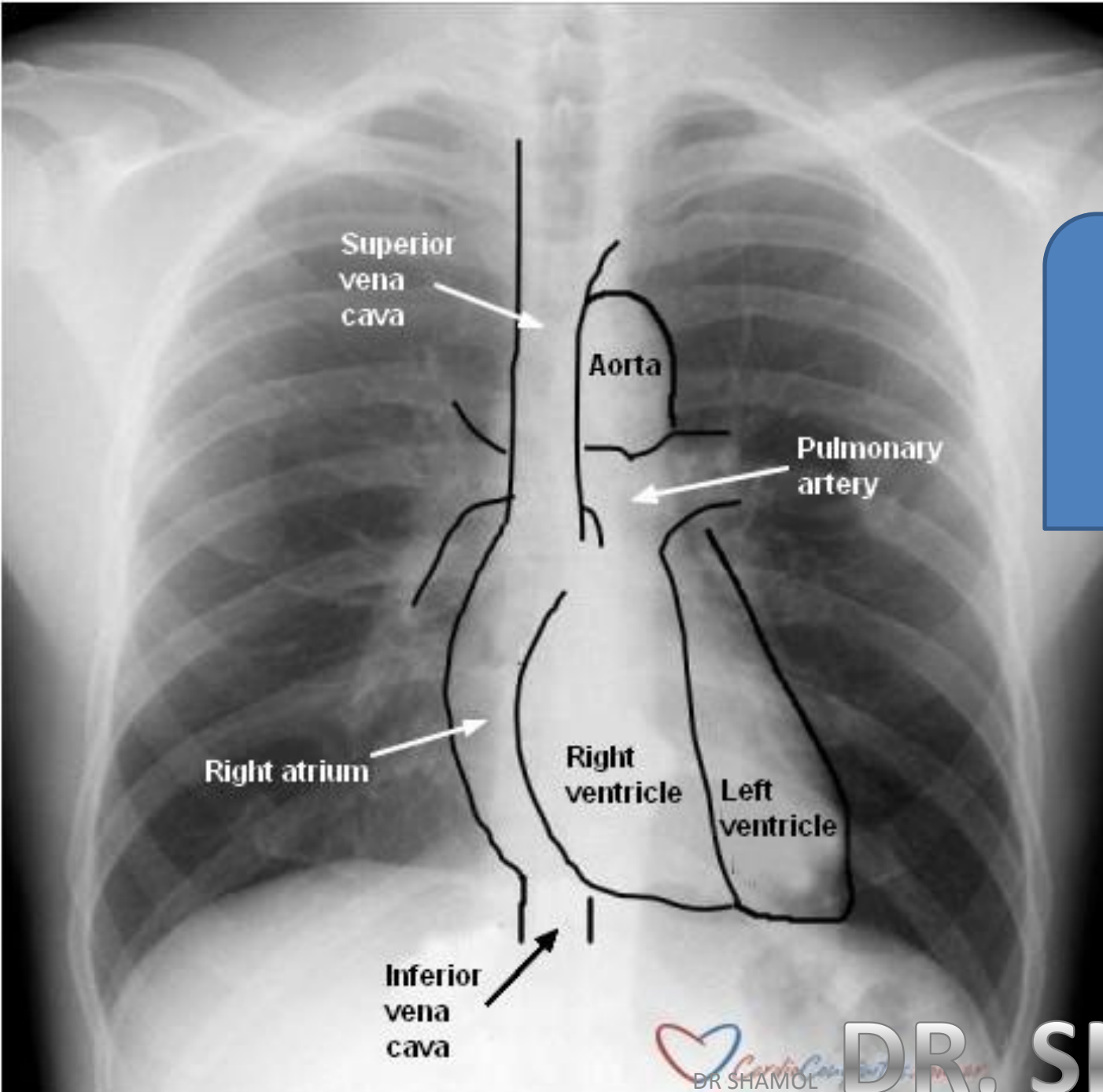
CXR-PA	from above to downward	upper lobe diversion of blood vessel
		Widening of carina.
		Left bronchus is horizontal
		fullness of pulmonary conus
		straightening of left border of heart
	central to periphery	Calcified shadow of mitral valve
		Double shadow in right border of heart
		Kerley's B lines
		Pulmonary edema
		Mottling or reticulonodular shadow due to pulmonary hemosiderosis.
	A---F & RBS	
	A—above	upper lobe diversion of blood vessel
	B—Bronchus	Left bronchus is horizontal
	C—carina	Widening of carina./calcified valve
	D--double	Double shadow in right border of heart
	E--edema	Pulmonary edema
	F-full	- fullness of pulmonary conus
	R—reticular	Mottling or reticulonodular shadow due to pulmonary hemosiderosis
	B—B-line	Kerley's B lines
	S-straightening	straightening of left border of heart

ECG	“P” is bifid (P-mitrale.)	
	May be RVH, RAH	
	bi-phasic p in V1	
	may have AF	
Echocardiography TEAR in MMC (nor for MBBS)	T-	<b>Thick mitral valve leaflet.</b>
	E-	<b>Enlarged</b> left atrium
	A-Area	valvular <b>area</b> decreased
	R-	<b>Reduced rate</b> of diastolic filing of left ventricle
	M	Characteristic “ <b>M</b> ” <b>shape movement</b> of anterior leaflt
	M	<b>Mural</b> thrombus
	C	<b>chordae tendinae</b> shorten
<b>Calcification of</b> valves (increased echogenecity).		
Doppler		
Cardiac catheter		

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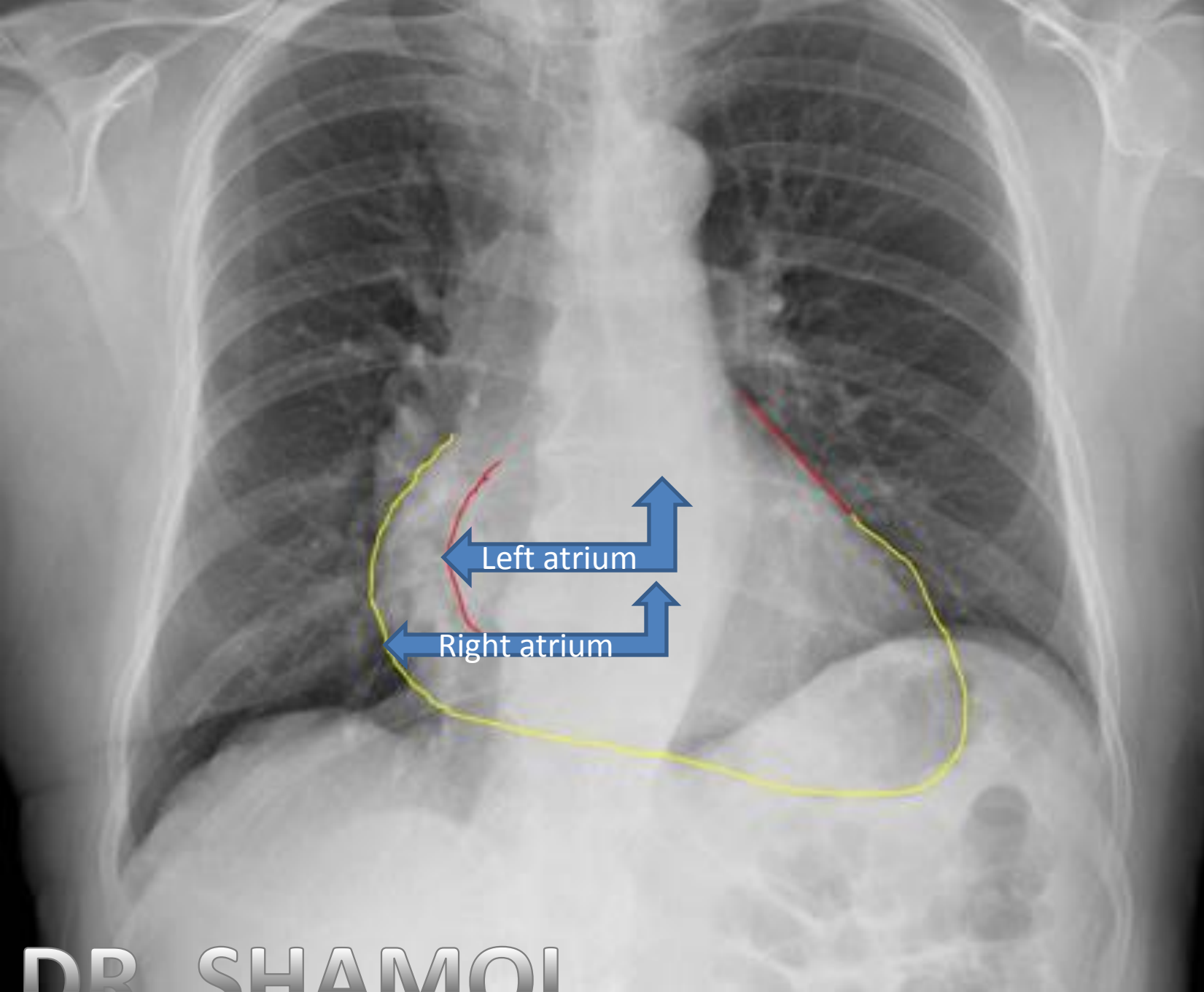


Normal  
X-ray



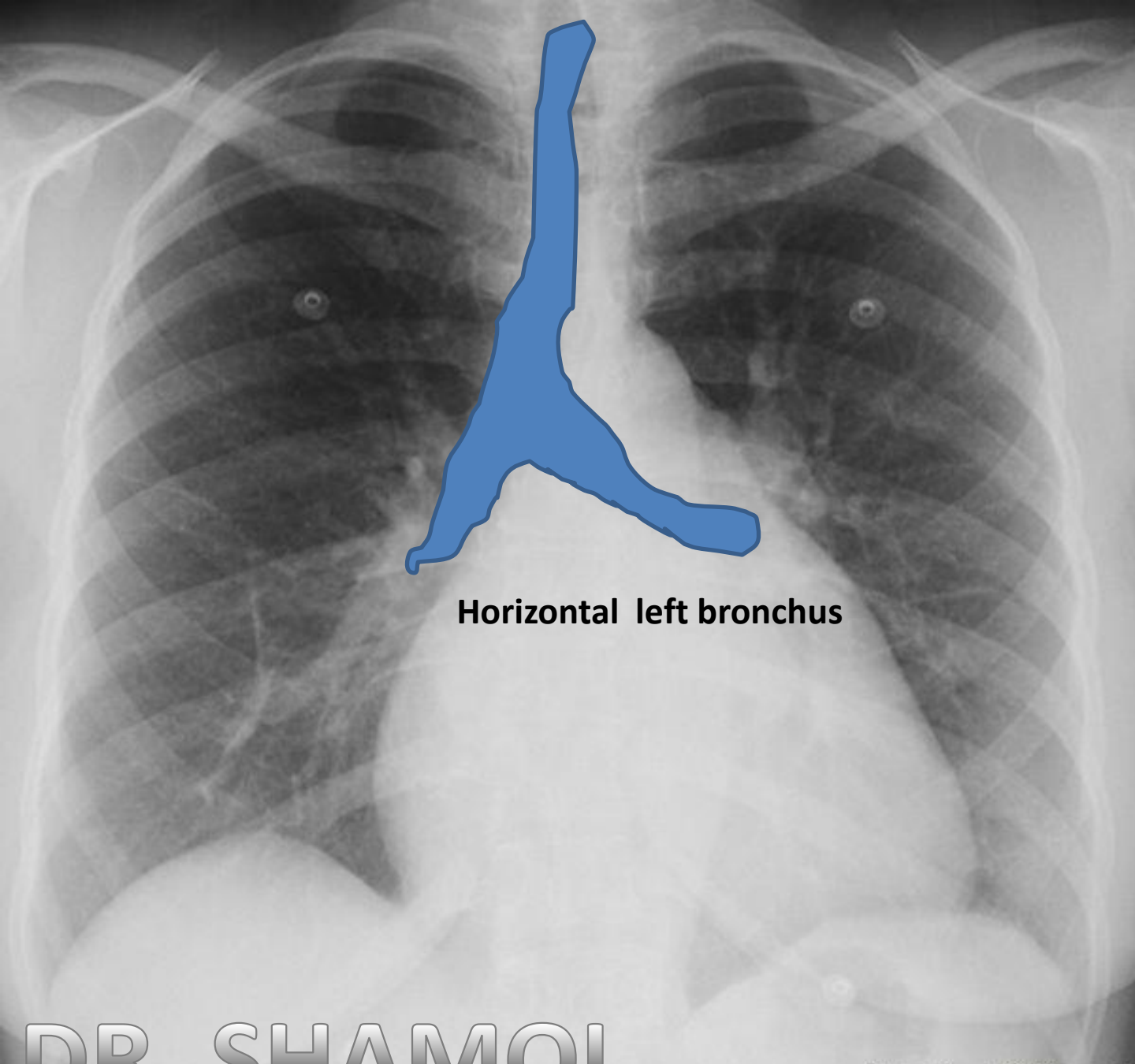
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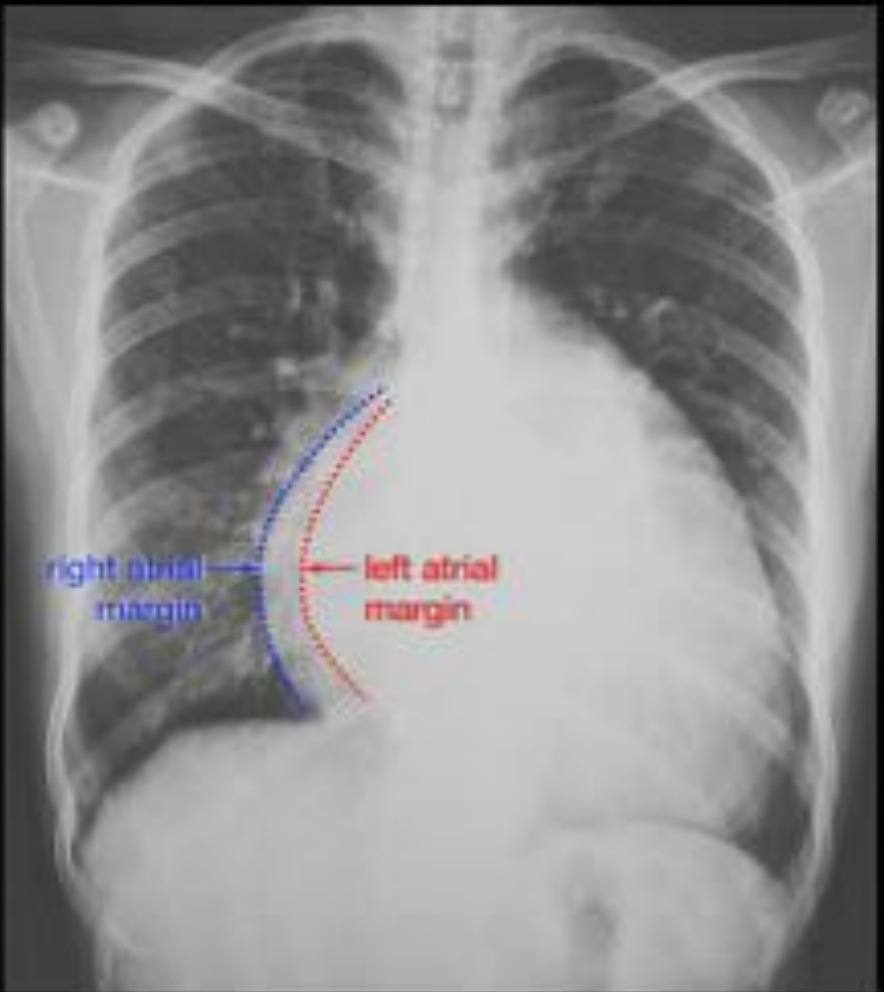


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**Horizontal left bronchus**



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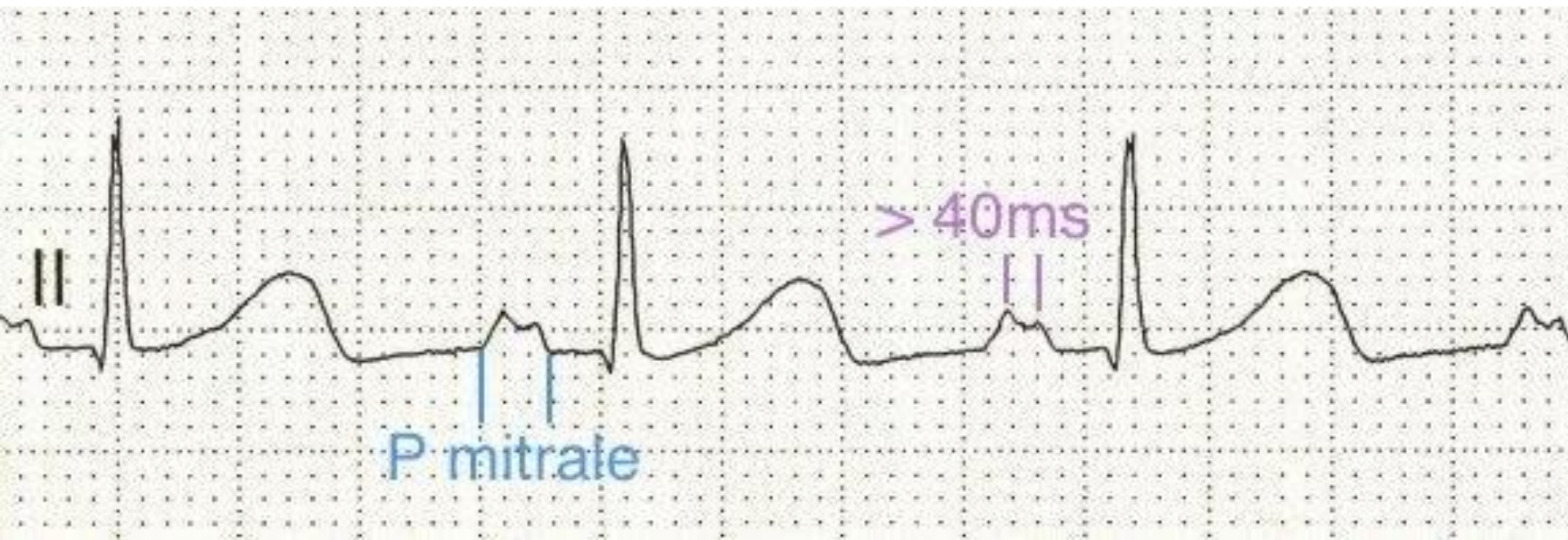
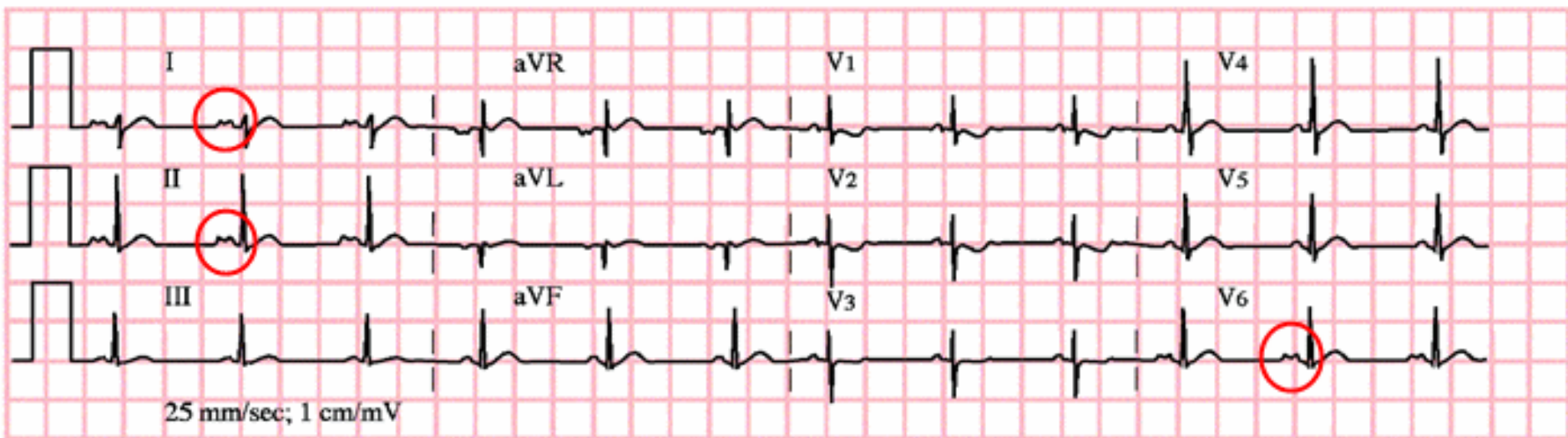




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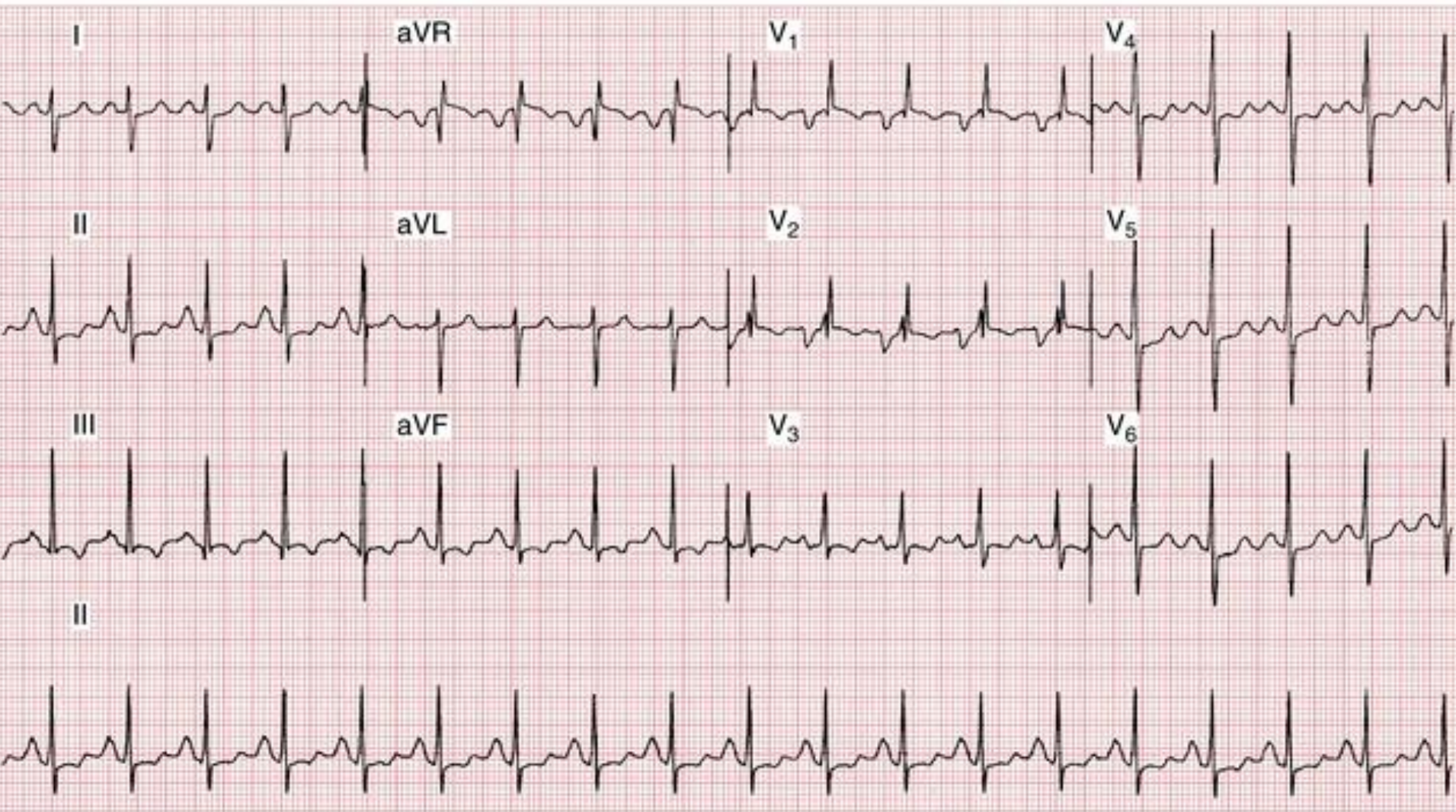


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# Severe Mitral Stenosis



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valve orifice	normal is 4 to 6 cm <sup>2</sup>	
	Severe when <1 cm <sup>2</sup> (tight ms).	
Signs of severe MS	P—Pulse	Pulse: Low volume
	O—onset	opening snap: nearer to the 2nd sound
	E— Evidence	Evidence of pulmonary hypertension and pulmonary congestion
	M—MDM	MDM: Prolonged
	S--Soft	1st heart sound: soft

What are the signs of **PH**

to remember pELLE	<b>P</b>	Palpable P2
	<b>E</b>	Epigastric pulsation (indicates RVH)
	<b>L</b>	Left parasternal heave (indicates RVH)
	<b>L</b>	Loud P
	<b>E</b>	Early diastolic murmur (know as Graham steel murmur due to pulmonary regurgitation)

What are the causes of mid diastolic murmur (MDM)?

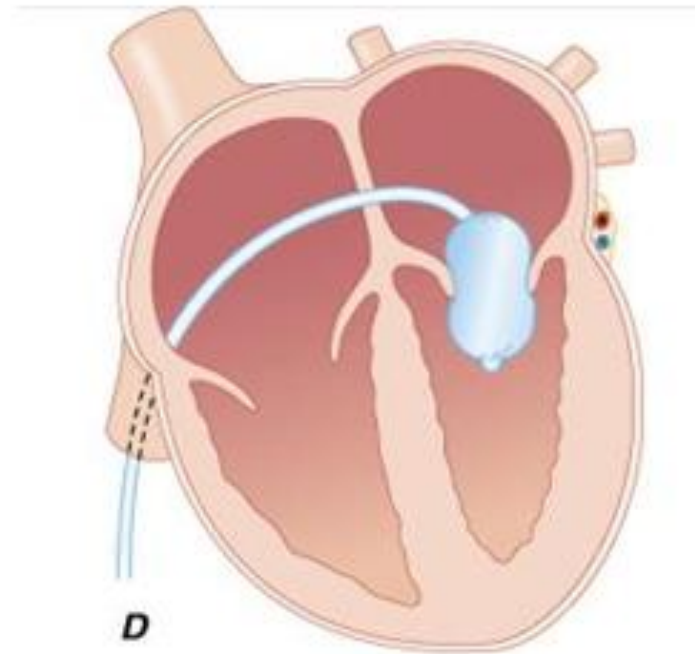
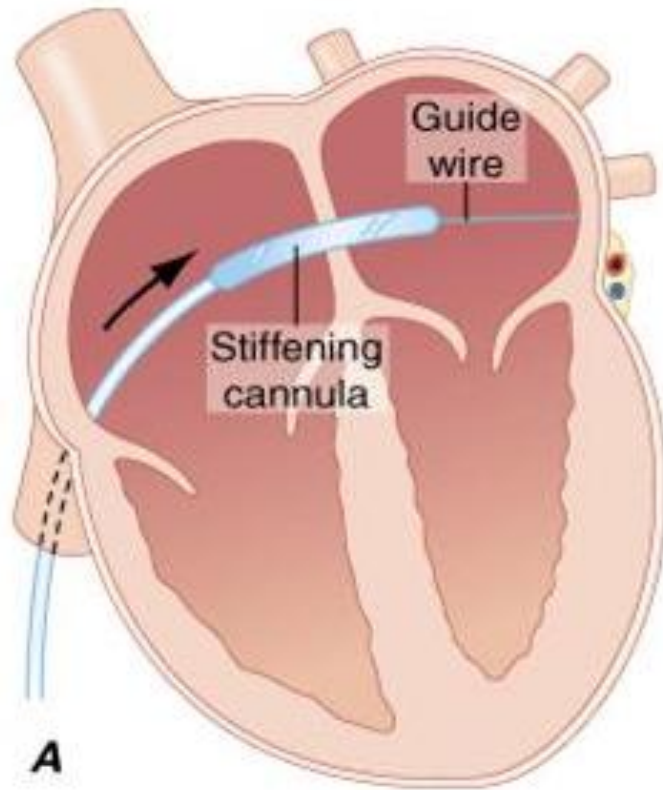
to remember MALTA-Car	<b>M</b>	mitral stenosis
	<b>A</b>	ASD (due to increased flow through tricuspid valve)
	<b>L</b>	Left atrial myxoma
	<b>T</b>	Tricuspid stenosis
	<b>A</b>	Austin-Flint murmur in aortic regurgitation
	<b>Car</b>	Carey Coomb's murmur (a soft MDM due to mitral valvulitis in acute rheumatic fever)



treatment	medical	Asymptomatic patient in sinus rhythm		follow up
		Mild symptoms:		diuretics to reduce left atrial pressure
				salt restriction
		If AF	rate control	B-blocker
				rate limiting calcium antagonists (e.g. verapamil, diltiazem).
				Digoxin
			Anticoagulant	
		CCF	Diuretic	
Antibiotic prophylaxis infective endocarditis is not routinely recommended				

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surgical treatment	type of RX	Valvotomy	CMC—closed mitral commissurotomy,
			OMC—open mitral commissurotomy
		Valvuloplasty	Treatment of choice (percutaneous balloon mitral valvuloplasty)
		Valve replacement	
	indication of valvuloplasty MINTS	M	<b>Mobile</b> , non-calcific valve/subvalve apparatus on echo
		I	<b>Isolated</b> mitral stenosis
		N	<b>No</b> (or trivial) mitral regurgitation
		T	LA free of <b>thrombus</b>
		S	Significant <b>symptoms</b>
	Indication of Valve replacement TMC	T	<b>Thrombus</b> in left atrium despite anticoagulation
		M	associated <b>MR</b>
		C	If the valve is <b>calcific</b> and rigid



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indication OF surgical treatment (NOT FOR MBBS) To remember smear pp	S	symptomatic moderate or severe MS when balloon valvuloplasty is unavailable
	M	MS MR/Moderate or severe MS with moderate or severe MR
	E	Episodes of pulmonary edema without precipitating cause
	A	Associated atrial fibrillation which does not respond to medical therapy
	R	Recurrent thromboembolism
	P	Pulmonary hypertension or recurrent hemoptysis
	P	pregnancy, with pulmonary edema
		as blood volume increases significantly with increased pulmonary pressure
		may be done in second trimester



patho-physiology	Rheumatic valvulitis → progressive fibrosis, calcification of the valve leaflets, and fusion of the cusps and subvalvular apparatus → results in a narrow, funnel-shaped orifice → The flow of blood from LA to LV is restricted → The pressure gradient between the LA and the LV increases → The LA gradually enlarges and hypertrophies → Pulmonary venous pressure rises → increase in pulmonary arterial (PA) pressure → results in right ventricular (RV) hypertrophy and RV enlargement → result in right atrial (RA) hypertension and enlargement → systemic venous congestion → tricuspid regurgitation
when PHTN occur	gradual rise in left atrial pressure → increase in pulmonary vascular resistance → Chronic changes in the pulmonary capillaries and pulmonary arteries include fibrosis and thickening → which leads to pulmonary hypertension → These changes prevent the lungs from the transudation of fluid into the alveoli → protect the patient from pulmonary oedema.
when pulmonary edema occur	in Atrial fibrillation → tachycardia and loss of atrial contraction lead to → rapid rise in left atrial pressure → precipitates pulmonary oedema
ATRIAL fibrillation	Atrial fibrillation due to progressive dilatation the LA
exercise and pregnancy	<p>exercise and pregnancy → Situations that increase demand → cardiac output → increase left atrial pressure</p> <p>increase in heart rate → shortens diastole → when the mitral valve is open → further rise in left atrial pressure → exercise intolerance</p>

What is the DD?	Tricuspid stenosis		
	Left atrial myxoma		
	Ball valve thrombus in left atrium,		
What this is not MDM murmur of ASD?			
causes of MDM in ASD	In atrial septal defect, large flow murmurs across the tricuspid valve can cause mid-diastolic murmurs		
clinical		ASD	MS
	1 <sup>st</sup> heart sound	not loud	loud
	splitting of second sound	wide, fixed splitting	no splitting
	opening snap	absent	present
ecg		incomplete right bundle branch block	p- mitralae `
What is Lutembacher syndrome?			
about 4% of the patients with atrial septal present in combination of MS this a combination called Lutembacher syndrome.			

<b>Difference between MS and TS</b>		<b>MS</b>	<b>TS</b>
	site	in mitral area left lateral position	murmur prominent in left lower parasternal edge
	respiration	increases after expiration	increases during inspiration
	other	loud 1 <sup>st</sup> heart sound	raised JVP
how will differentiate from left atrial myxoma		<b>In left atrial myxoma</b>	<b>MS</b>
	relation with posture	murmur change with posture	no relation with posture
	systemic feature	history of fever, weight loss, myalgia, arthralgia, skin rash, Raynaud's	not present

how will differentiate from Austin-Flint murmur		Austin-Flint murmur	MS
	peripheral sign of AR, collapsing	present	absent
	loud 1 <sup>st</sup> heart sound opening snap	absent	present
Causes of haemoptysis in MS?	Haemoptysis can occur for a variety of reasons		
	pulmonary oedema	alveolar capillary rupture → pink frothy	
	pulmonary hypertension	Rupture of pulmonary or bronchial veins associated with → frank hemoptysis	
	pulmonary infarction		
	blood-stained sputum of chronic bronchitis		

### Why there is pulmonary hypertension in MS?

Because of:

Passive backward transmission of raised left atrial pressure

Reflex pulmonary artery vasoconstriction

Organic obliterative change in pulmonary vascular bed



<b>If the patient with MS suddenly becomes unconscious, what is the likely cause?</b>
CVD (cerebral infarction), usually with right sided hemiplegia. Usually CVD occurs when there is associated atrial fibrillation (AF).
<b>What may be the cause of CVD in this case?</b>
Cerebral embolism (involving lenticulostriate branch of the left middle cerebral artery, causing infarction of the internal capsule).
<b>Why syncope may occur in MS?</b>
. Due to reduction of cardiac output. Also, may be due to atrial fibrillation with fast ventricular rate, pulmonary hypertension, pulmonary embolism, ball valve thrombus, cerebral embolism.
<b>What is paroxysmal nocturnal dyspnea (PND)?</b>
Paroxysmal breathlessness usually at night, which awakens the patient from sleep with a feeling of suffocation and choking sensation. The patient sits, stands up or open the windows for fresh air. It is due to pulmonary edema, and indicates early LVF or left atrial failure
<b>What are the causes of PND?</b>
<ul style="list-style-type: none"> <li>• Venous return increases while lying down</li> <li>• Mobilization of edema fluid from extravascular to intravascular compartment in supine position.</li> <li>• Low adrenergic drive at sleep.</li> <li>• Heart rate increases during rapid eye movement (REM) sleep.</li> <li>• Vital capacity is reduced in supine position.</li> <li>• Left atrial pressure is elevated and PaO<sub>2</sub> is reduced during sleep.</li> <li>• abdominal contents are also pushed up giving pressure on diaphragm</li> </ul>

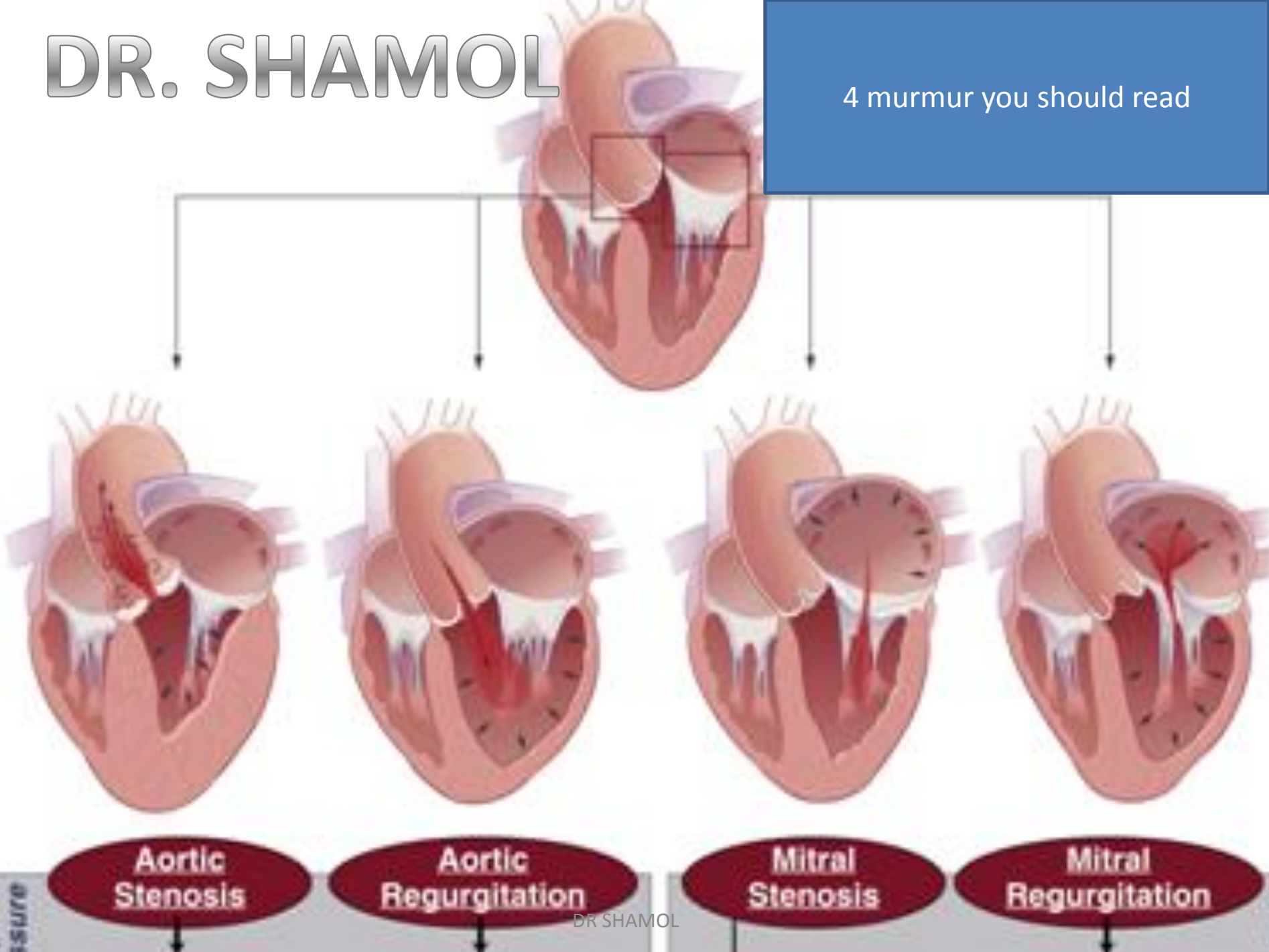
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PRegnAnCy	cardiac output demands on the heart increase by 70%		
	symptomatic status follows the rule of one class	the patient's symptoms will increase by 1 New York Heart Association class during pregnancy	
		asymptomatic patient may develop class II symptoms,	
		whereas a class II patient may become class III	
	pt symptomatic with NYHA class III before pregnancy	avoid pregnancy	
		do BMV (ballon mitral valvoplasty )	
	commonest complication	pulmonary oedema, secondary to ↑ left atrial pressure precipitated by	increased heart rate
			or ↑ volume (such as occurs during the third stage of labour or following injudicious intravenous fluid therapy

treatment	advise BMV before conception	moderate to severe symptoms	
		severe stenosis (MV area $<1\text{ cm}^2$ )	
		NYHA class III	
		if the valve is not amenable to valvotomy, wait until after MV replacement	
	mild symptoms	Diuretics should be continued in pregnancy	
	balloon mitral valvotomy	if medical therapy fails, or,	
		for those with severe MS	
		done in 2 <sup>nd</sup> trimester of pregnancy	
	if Pulmonary oedema,	oxygen and diuretics	
		introduction or reintroduction of a B-blocker to slow heart rate	
	Labour	Women with MS should avoid the supine and lithotomy positions as much as possible for labour and delivery	
		Fluid overload must be avoided, and even in the presence of oliguria, Without significant blood loss, the temptation to give intravenous fluids must be resisted.	
	AF	a $\beta$ -blocker to slow heart rate	
		issue of anticoagulation is highly controversial	
		warfarin-- during the first trimester is contraindicated due to high risk of embryopathy	
		risk of stroke increased	
		transesophageal echo	if no atrial thrombus $\rightarrow$ DC cardioversion
		If thrombus $\rightarrow$ Use of enoxaparin	

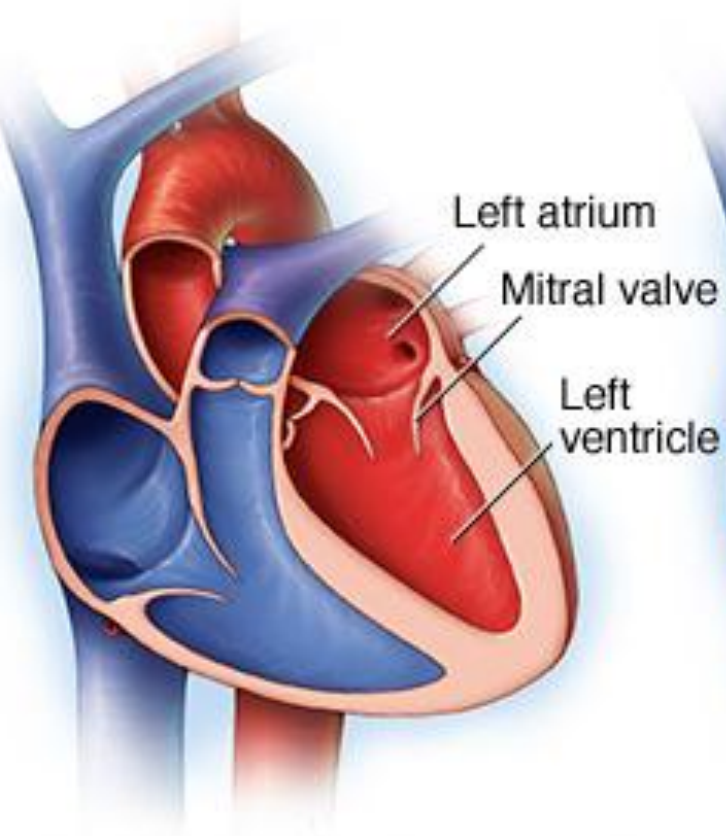
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4 murmur you should read

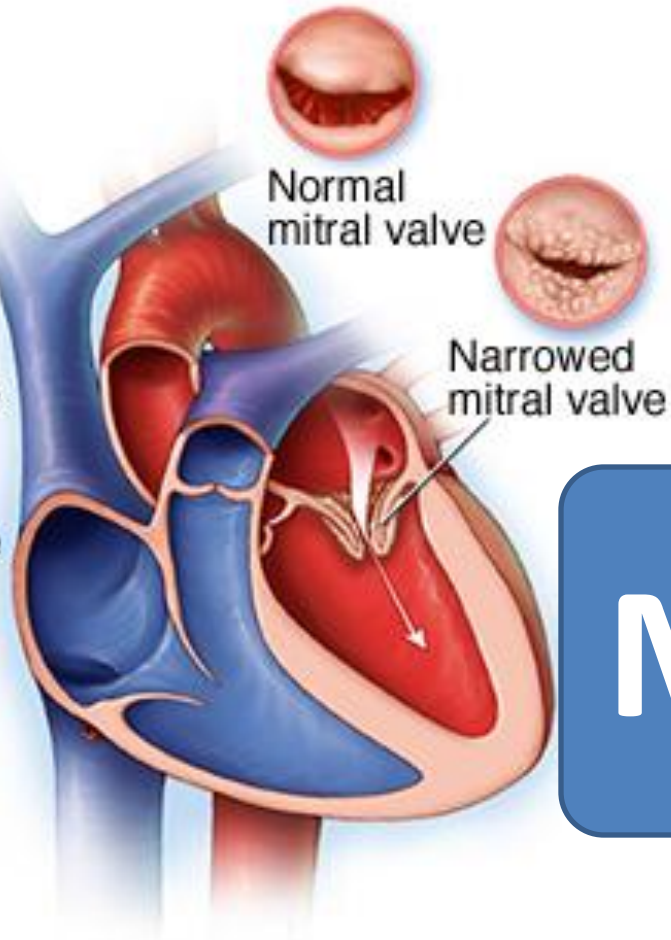




Normal heart



Mitral valve stenosis



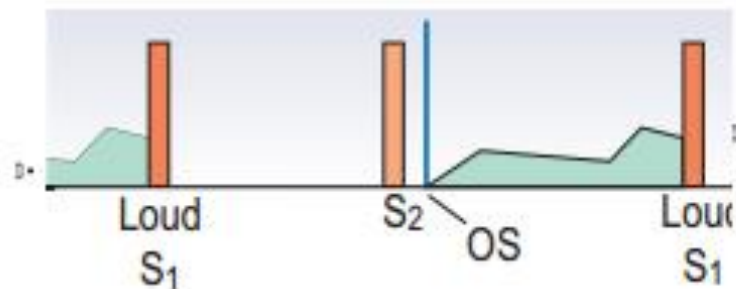
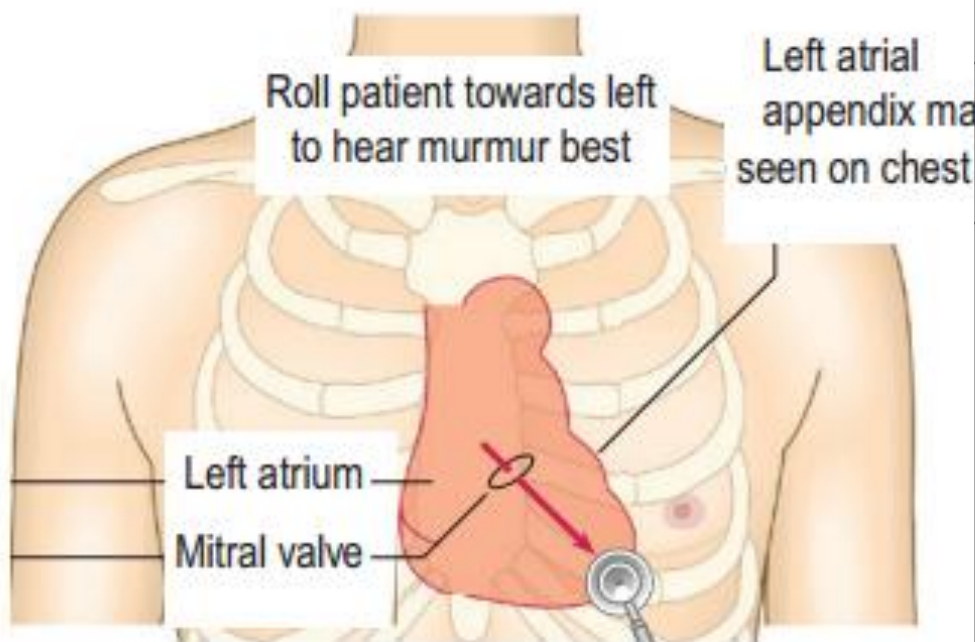
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apex beat : Tapping & not shifted

first heart sound: Loud  
( Tell it variable intensity if AF)

Opening snap

Murmur

:Low pitched , localized , rough ,  
rumbling Mid diastolic murmur ,  
with pre systolic accentuation

❖ Best heard with bell of  
stethoscope in left lateral position  
and breath hold expiration

❖ Presystolic accentuation --  
absent if associated AF

❖ Opening snap –absent if valve is  
heavily calcified

If patient developed pulmonary  
hypertension then you find following

- ❖ palpable P2
- ❖ Left para-sternal heave

Auscultation

- ❖ loud P2

## What are the complication of MS?

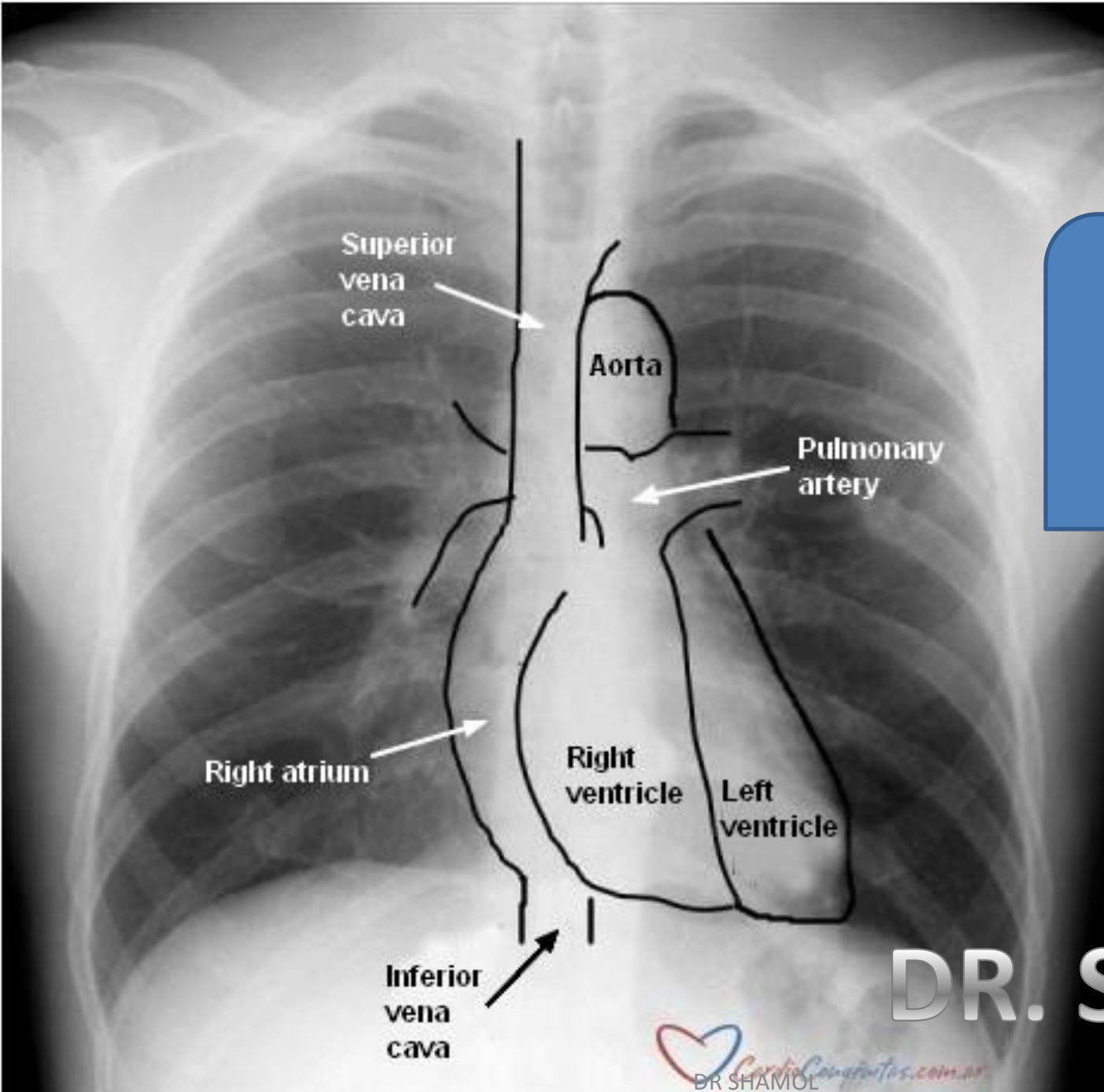
Firs utter the following 4 cause then other s (CASE)-history

(A)	Atrial fibrillation
(S)	Stroke /Systemic Thrombo-embolism
(C)	Right heart failure or CCF (due to pulmonary hypertension )
(E)	pulmonary edema
<b>H/ History</b>	pulmonary hypertension

Signs of severe MS	P—Pulse	Pulse: Low volume
	O—onset	opening snap: nearer to the 2nd sound
	E— Evidence	Evidence of pulmonary hypertension and pulmonary congestion
	M—MDM	MDM: Prolonged
	S--Soft	1st heart sound: soft

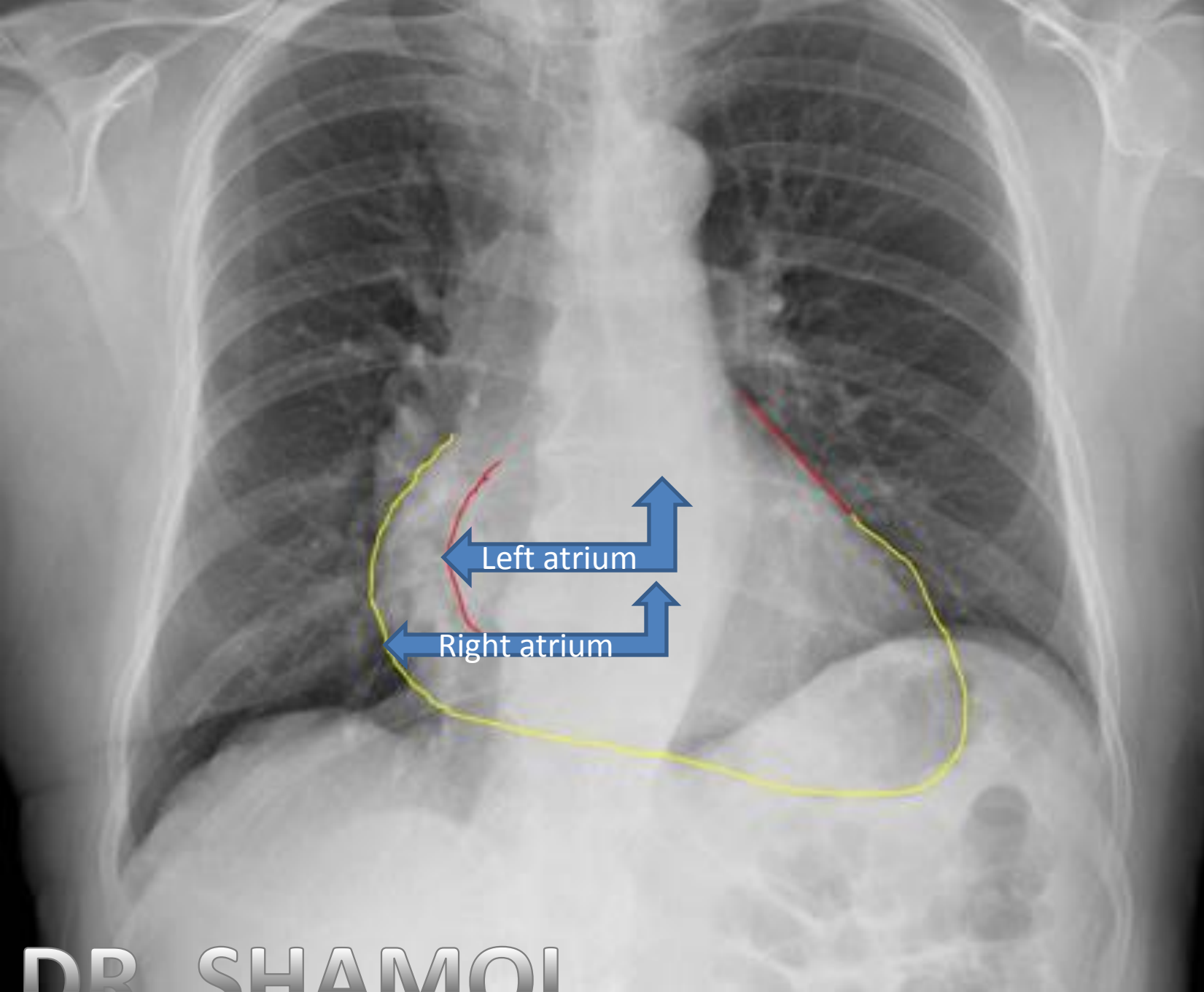
ECG	“P” is bifid (P-mitrale.)	
	May be RVH, RAH	
	bi-phasic p in V1	
	may have AF	
Echocardiography TEAR in MMC (nor for MBBS)	T-	<b>Thick mitral valve leaflet.</b>
	E-	<b>Enlarged</b> left atrium
	A-Area	valvular <b>area</b> decreased
	R-	<b>Reduced rate</b> of diastolic filing of left ventricle
	M	Characteristic “ <b>M</b> ” <b>shape movement</b> of anterior leaflet
	M	<b>Mural</b> thrombus
	C	<b>chordae tendinae</b> shorten <b>Calcification</b> of valves (increased echogenecity).
Doppler		
Cardiac catheter		





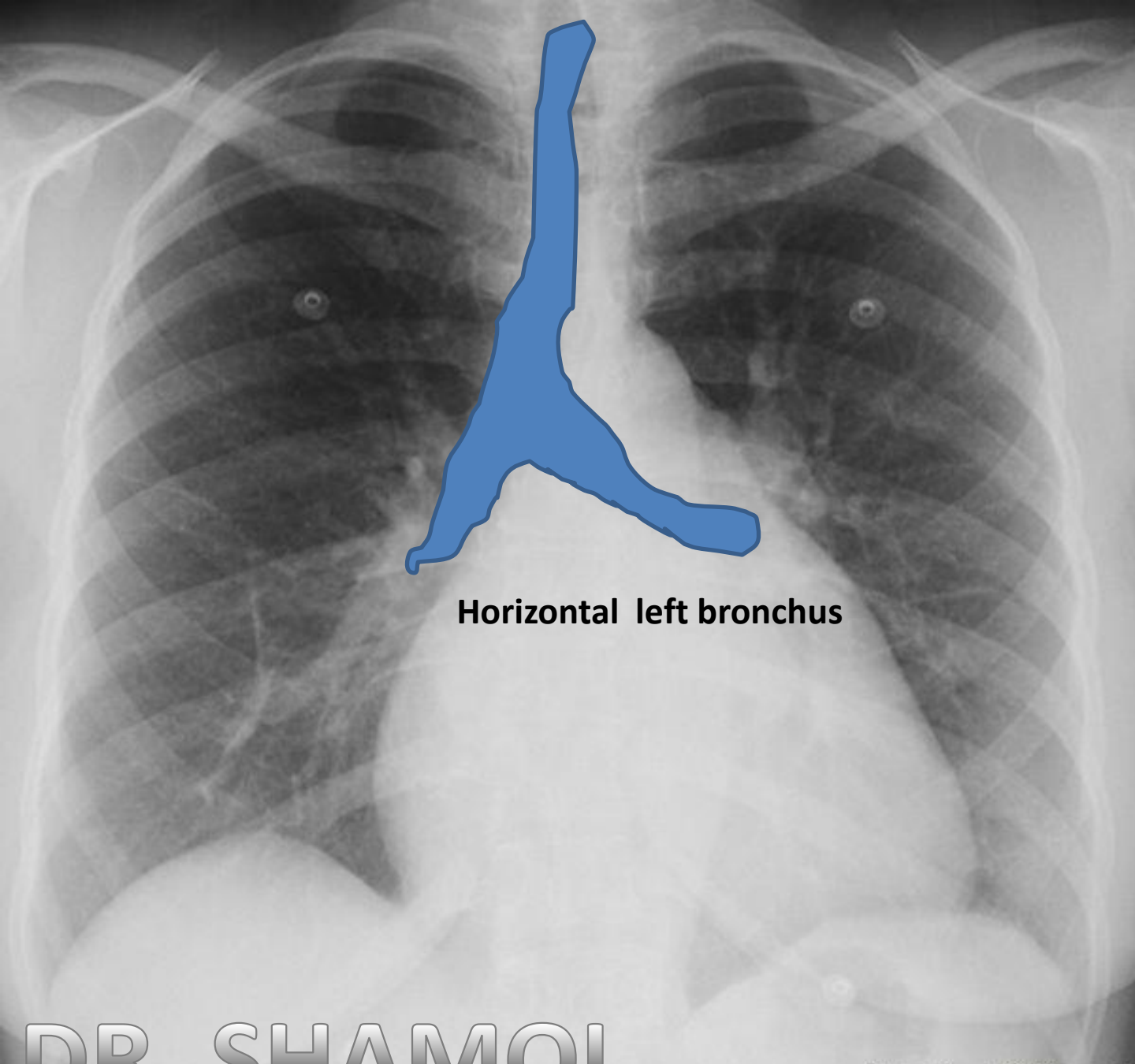
Normal  
X-ray

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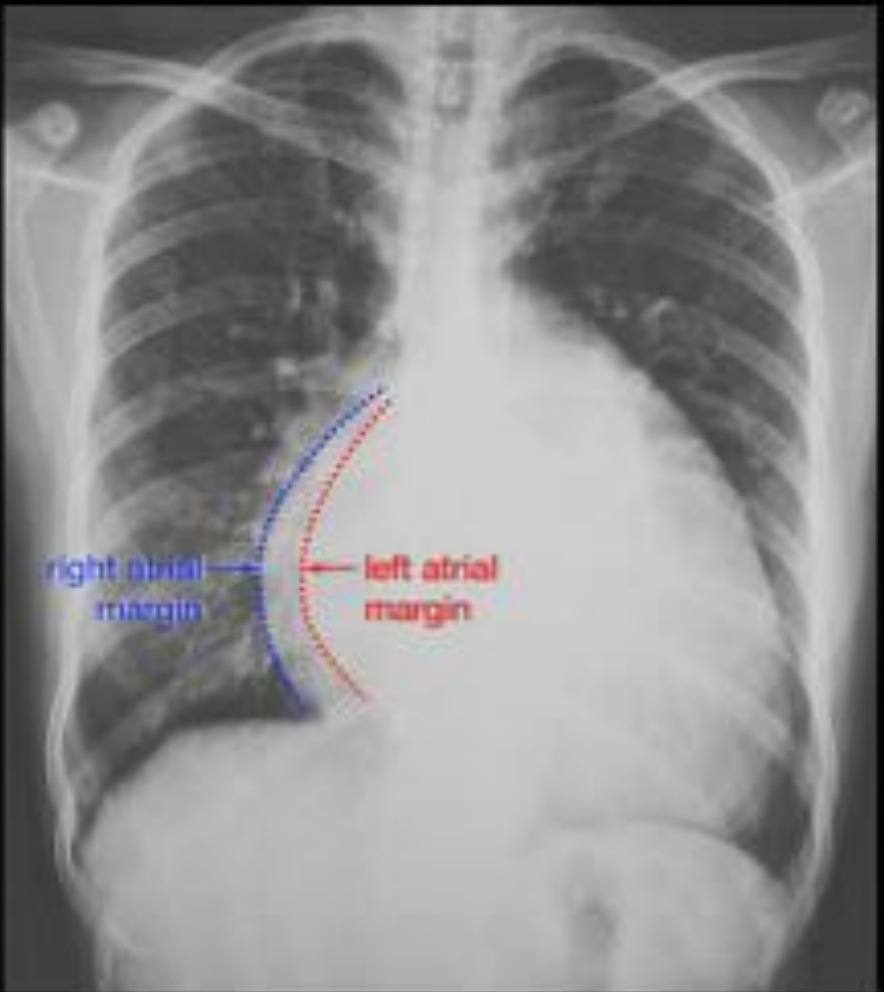


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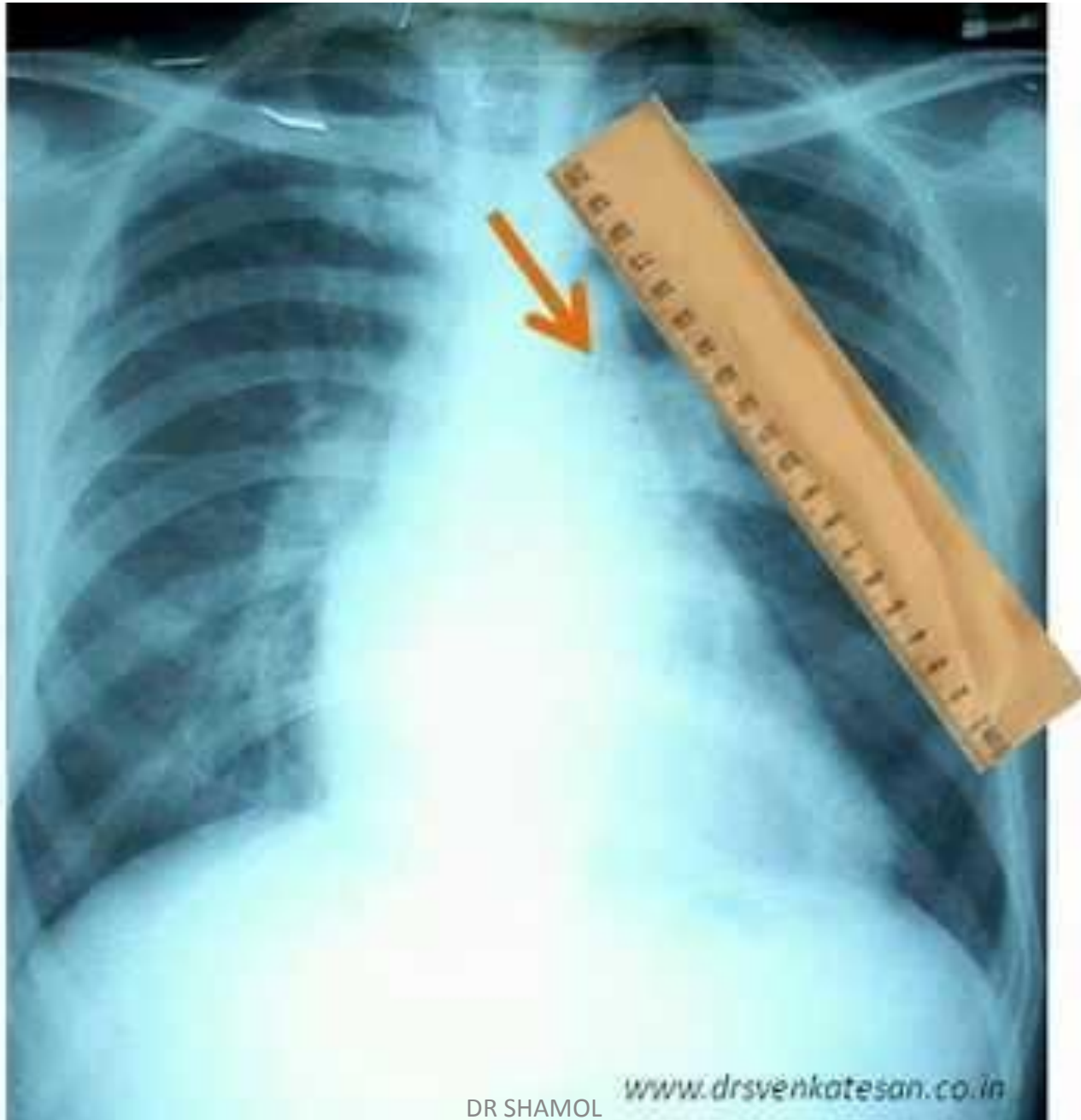
**Horizontal left bronchus**

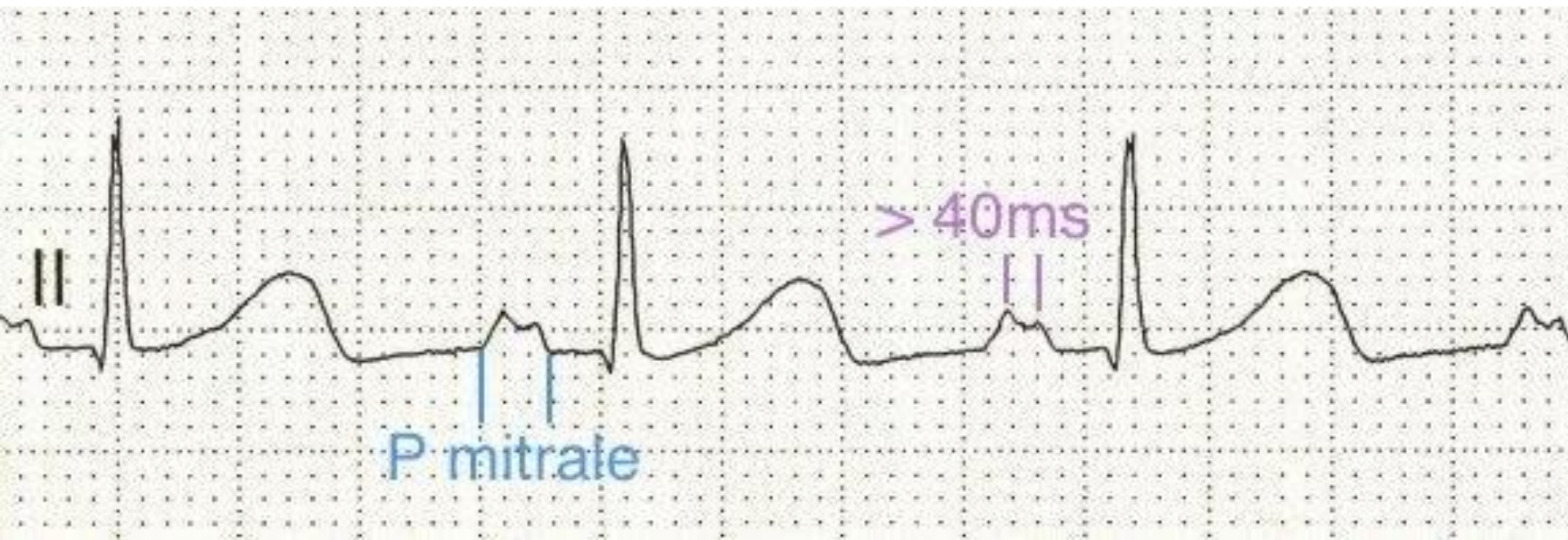
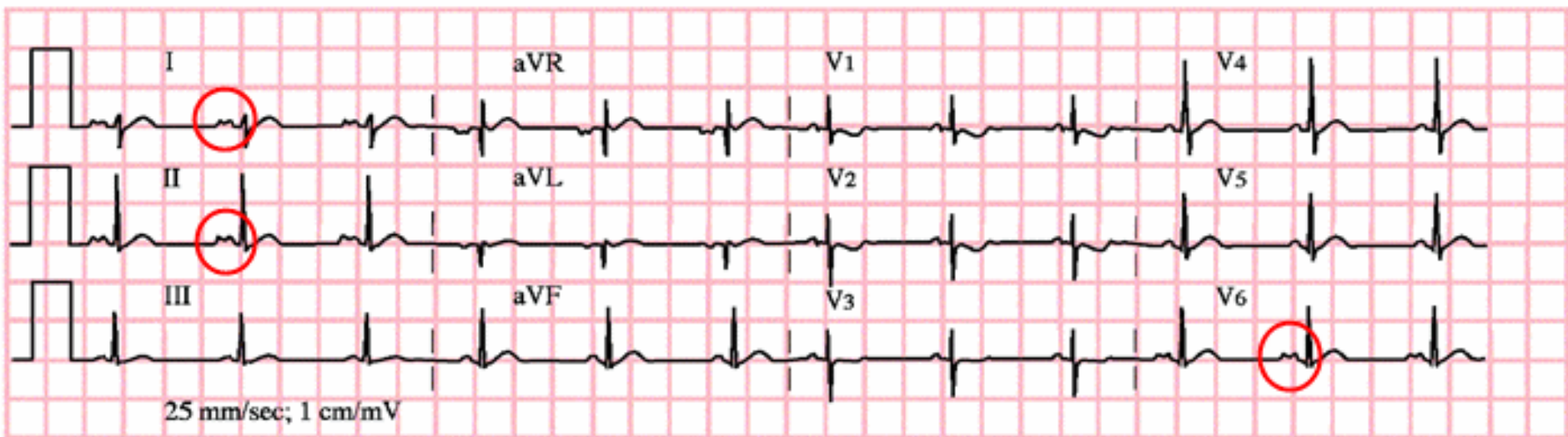


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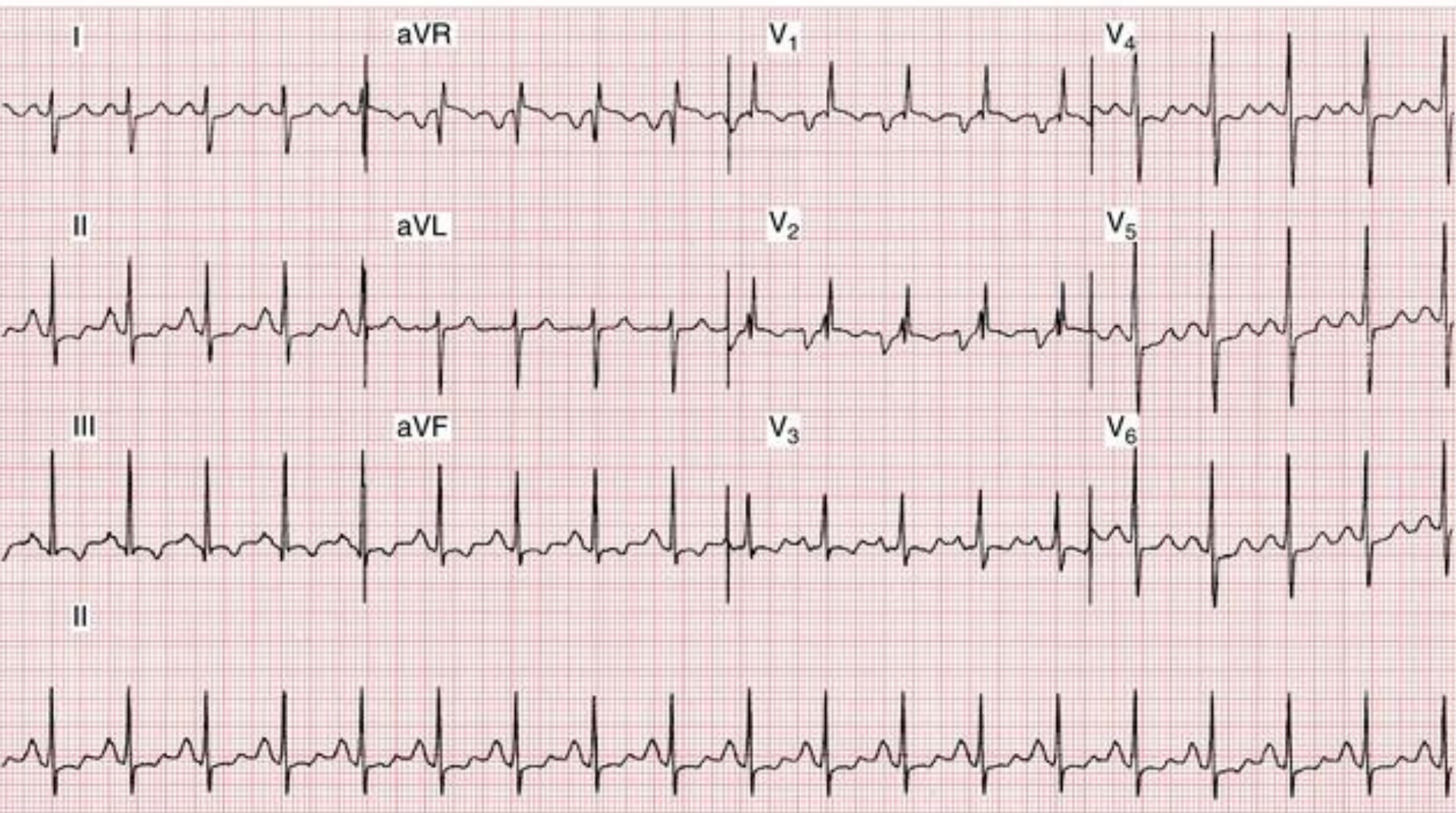


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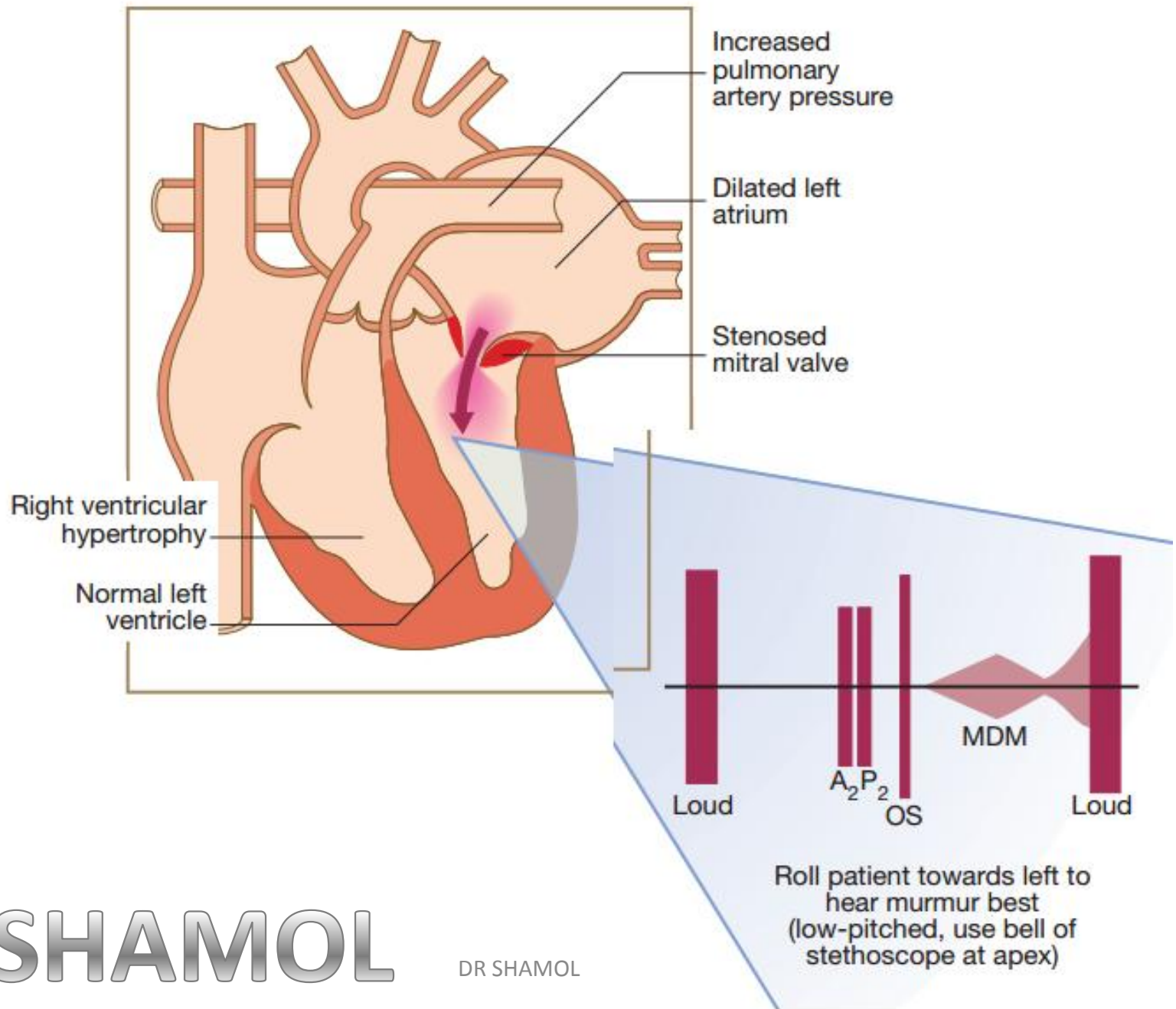
# Severe Mitral Stenosis



treatment	medical	Asymptomatic patient in sinus rhythm		follow up
		Mild symptoms:		diuretics to reduce left atrial pressure
				salt restriction
		If AF	rate control	B-blocker
				rate limiting calcium antagonists (e.g. verapamil, diltiazem).
				Digoxin
			Anticoagulant	
		CCF	Diuretic	
Antibiotic prophylaxis infective endocarditis is not routinely recommended				

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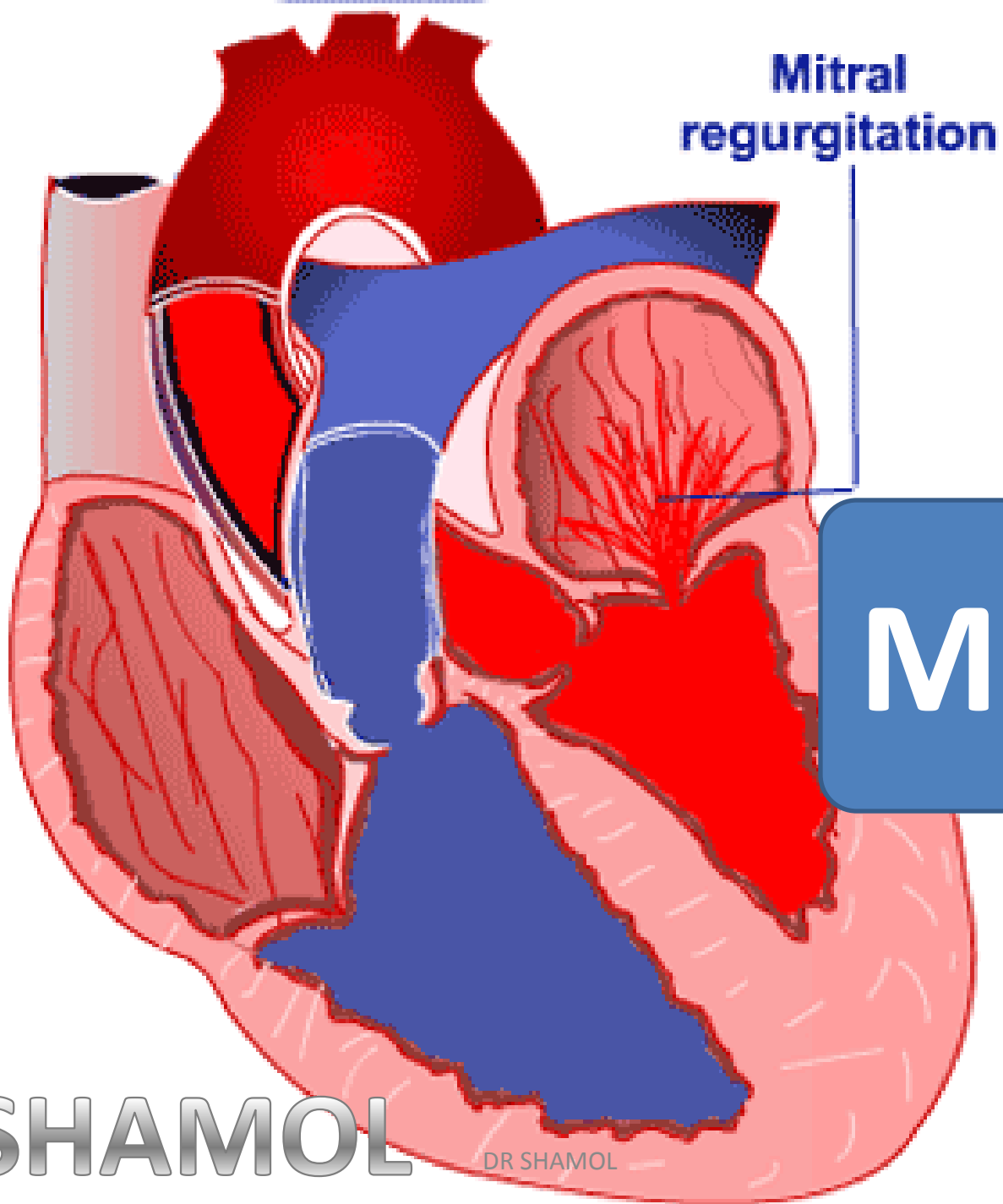




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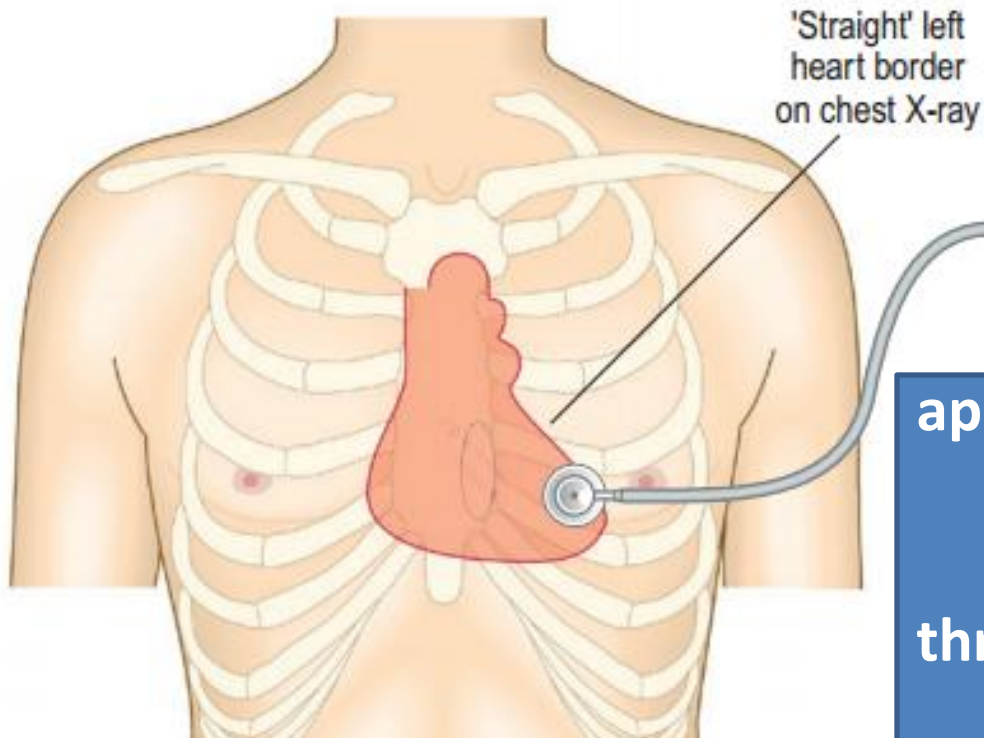
surgical treatment	type of RX	Valvotomy	CMC—closed mitral commissurotomy,
			OMC—open mitral commissurotomy
		Valvuloplasty	Treatment of choice (percutaneous balloon mitral valvuloplasty)
		Valve replacement	
	indication of valvuloplasty MINTS	M	<b>Mobile</b> , non-calcific valve/subvalve apparatus on echo
		I	<b>Isolated</b> mitral stenosis
		N	<b>No</b> (or trivial) mitral regurgitation
		T	LA free of <b>thrombus</b>
		S	Significant <b>symptoms</b>
	Indication of Valve replacement TMC	T	<b>Thrombus</b> in left atrium despite anticoagulation
		M	associated <b>MR</b>
		C	If the valve is <b>calcific</b> and rigid



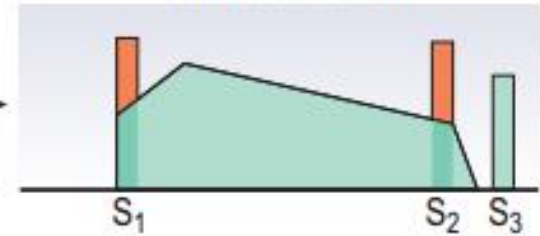
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Apical pansystolic murmur  
radiates to axilla



**apex beat :**

shifted and thrusting in  
nature

**thrill :**

systolic thrill at apex.

**Heart sound :**

soft first heart sound

**MURMUR:**

Loud ,blowing Pansystolic  
murmur .Best heard at the  
apex radiate to axilla

If patient developed pulmonary  
hypertension then you find  
following

- ❖ palpable P2
- ❖ Left para-sternal heave

**Auscultation**

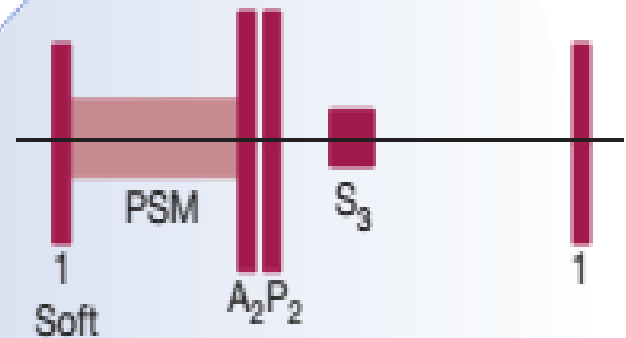
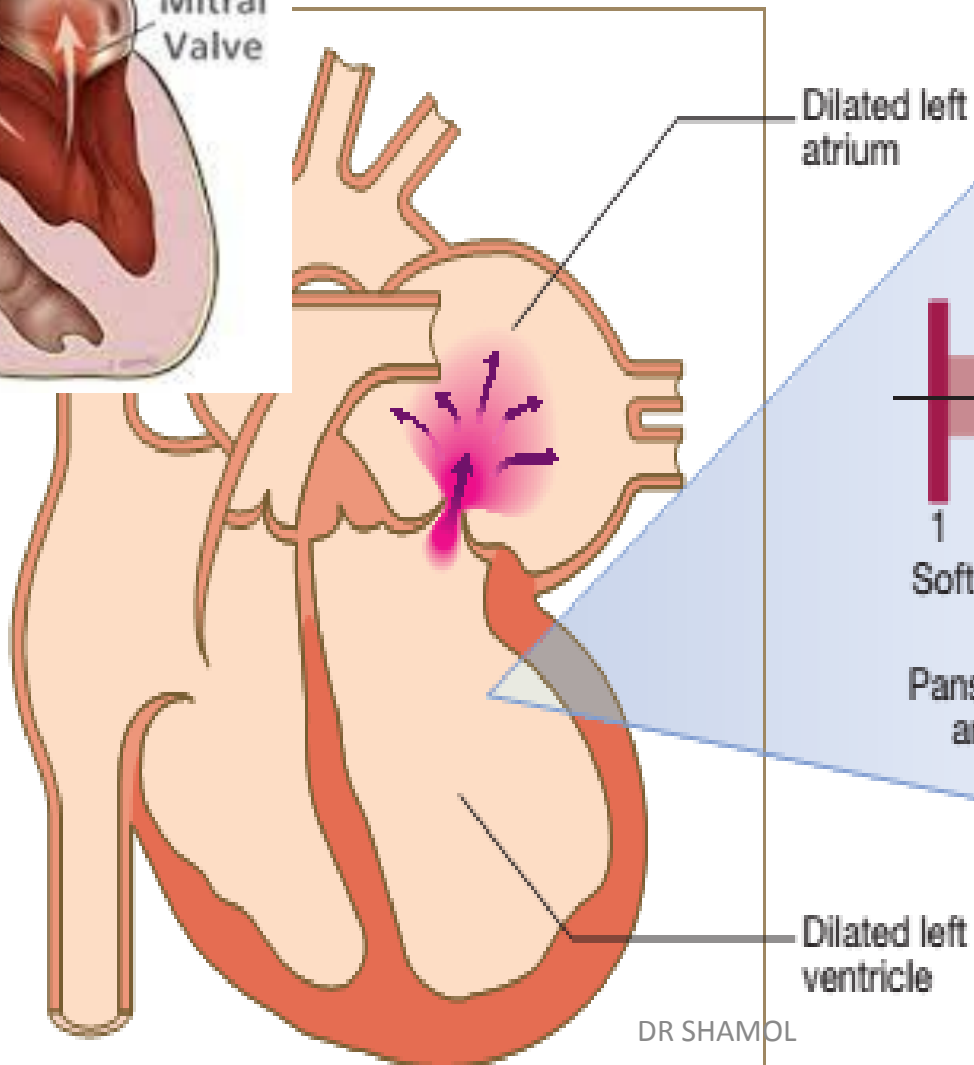
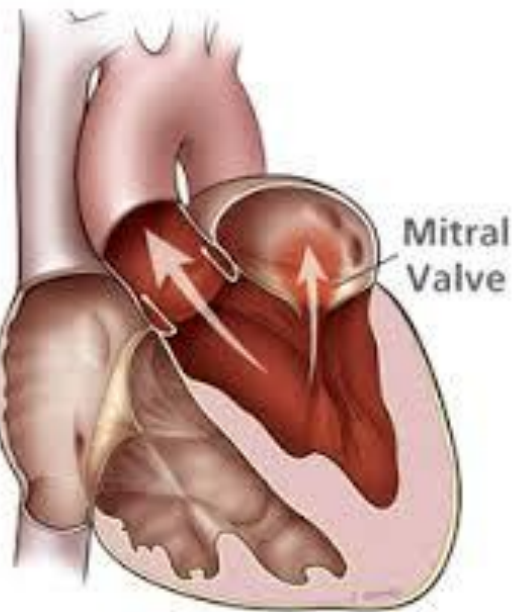
- ❖ loud P2



treatment		
<b>mild to moderate case</b>	Diuretic	
	Vasodilators-- ACE inhibitors	
	AF	If fast rate : Digoxin
		Anticoagulants
	prophylaxis against endocarditis not routinely recommended	
	Follow-up	Every 6 months by echocardiogram.
valve replacement considered	If the ejection fraction falls to 55%	
	and left ventricular dilatation >60 mm	
	In severe MR or in progressively worsening MR	
<b>acute severe MR</b>	require urgent stabilization and preparation for surgery	
	Diuretics	
	intravenous vasodilators particularly sodium nitroprusside	
	even intraaortic balloon counterpulsation	
.Rx of MR due to coronary artery disease	undergo coronary bypass graft surgery, plus	
	repair the valve and restore mitral valve function by inserting an annuloplasty ring to overcome annular dilatation and to bring the valve leaflets closer together	

symptoms	Symptoms depend on how suddenly the regurgitation develops	
	sudden-onset mitral regurgitation usually presents with acute pulmonary oedema	
chronic case FOPD	<b>F</b> -- Fatigue	due to low cardiac output
	<b>O</b> -- Oedema, ascites	due to right heart failure
	<b>P</b> -- Palpitation	due to atrial fibrillation, increased stroke volume
	<b>D</b> -- Dyspnoea	due to pulmonary venous congestion
complications of MR	L	Acute LVF
	A	Arrhythmia (atrial fibrillation, ectopics)
	S	Stroke / embolism
	I	Infective endocarditis
	C	CCF.
signs of severe MR LFT	L-LV	Large left ventricle (apex is shifted, thrusting)
	F-flow murmur	Presence of mid diastolic flow murmur (due to rapid filling of the left ventricle).
	T-Third	Presence of third heart sound

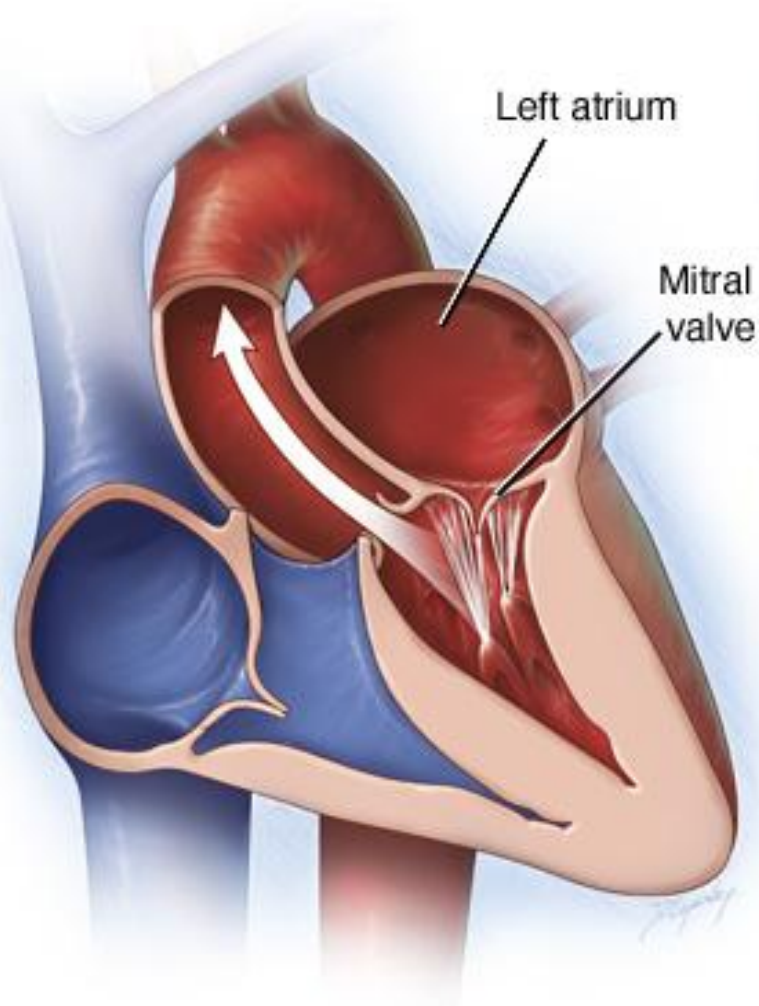
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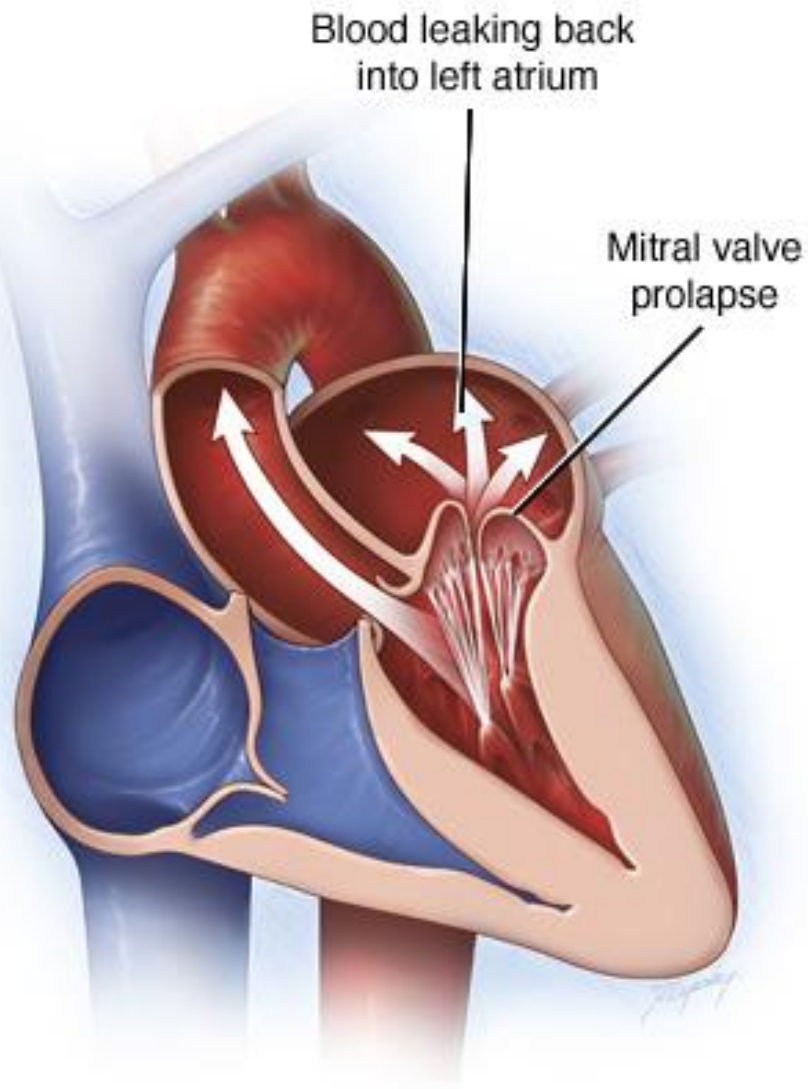
Pansystolic murmur heard best at apex and left sternal edge (diaphragm), radiates to axilla

causes			
acute MR <b>MISTAR</b>	M	Acute myocardial infarction (due to rupture of papillary muscle)	
	I	Infective endocarditis (due to perforation of mitral valve leaflet or chordae)	
	S	surgery (mitral valvotomy)	
	T	Trauma	
	A	Acute rheumatic fever (due to mitral valvulitis)	
	R	Spontaneous rupture of chorda tendineae or myxomatous degeneration of valve	
chronic MR to remember <b>MRCP -TIA</b>	M	Mitral valve prolapsed( myxomatous degeneration)	
	R	Chronic rheumatic heart disease	
	C	Connective tissue diseases	RA, SLE,
			Marfan's syndrome,
			Ehler-Danlos syndrome
		Cardiomyopathy → Secondary to left ventricular dilatation— ICM, DCM, Hypertrophic obstructive cardiomyopathy (HOCM)	
	P	Papillary muscle dysfunction due to acute myocardial infarction	
	T	Trauma or mitral valvotomy	
	I	Infective endocarditis	
	A	ankylosing spondylitis	





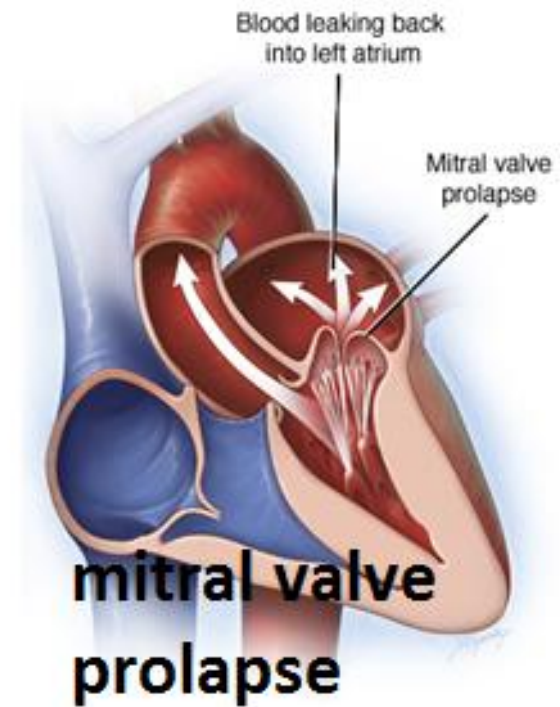
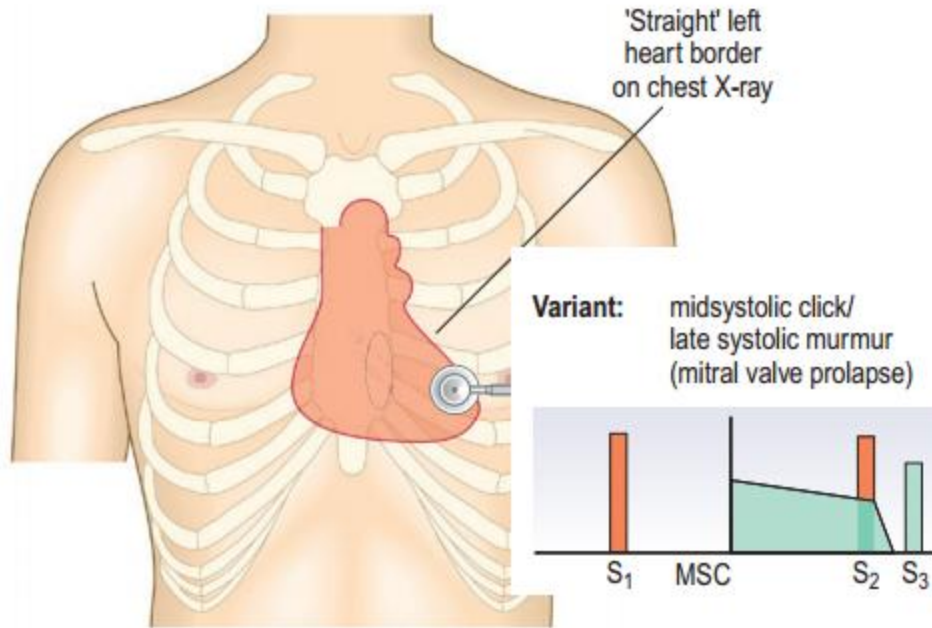
Normal heart



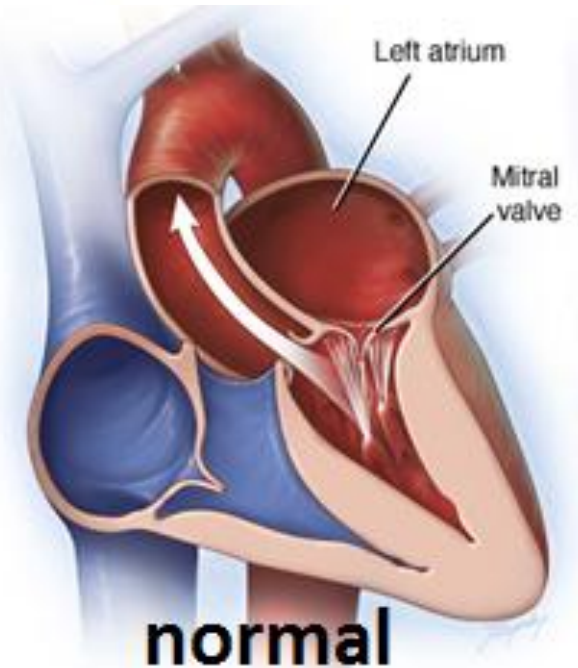
Mitral valve prolapse  
with regurgitation

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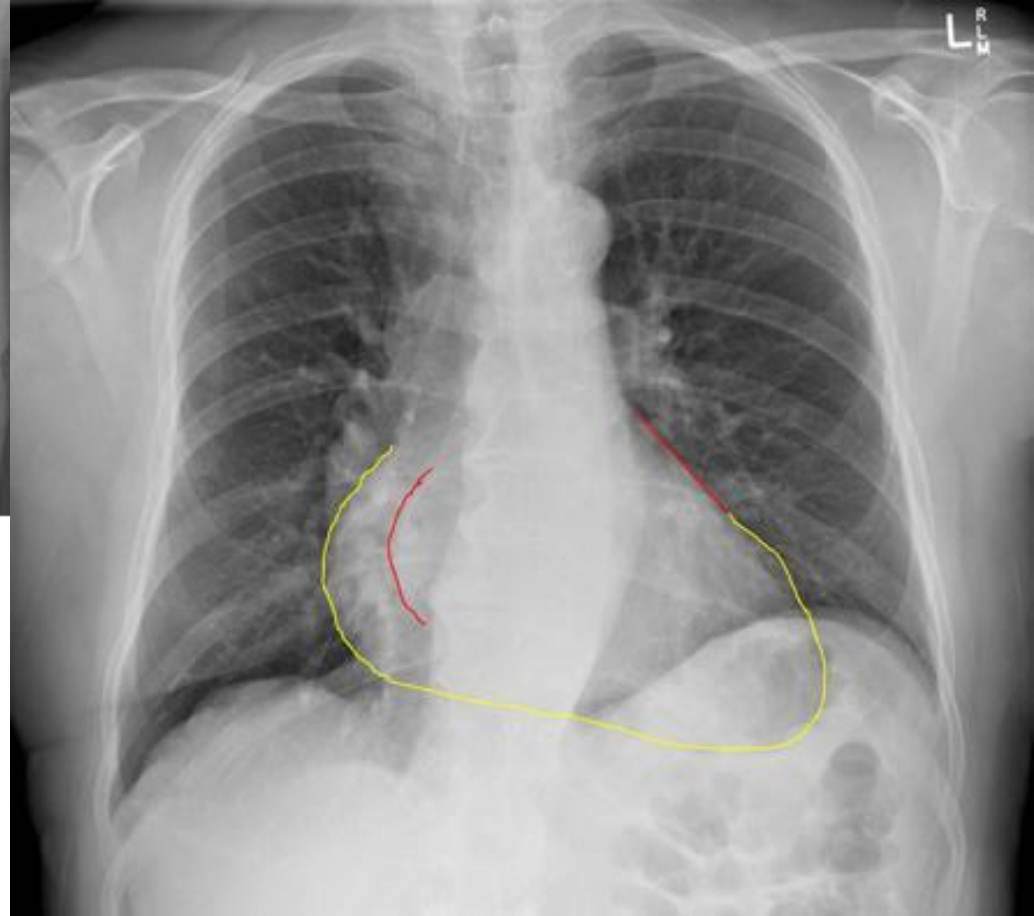
In **mitral valve prolapse**:  
there is a mid-systolic click (MSC) followed by mid or late systole murmur



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<b>ECG</b>	Left atrial hypertrophy/ broad bifida P waves (P mitrale)
	Left ventricular hypertrophy
	if the underlying causes is coronary artery disease ECG change of inferior or posterior wall myocardial infarction seen
<b>CXR -PA</b>	cardiomegaly (Enlarged LA, Enlarged LV)
	Pulmonary venous congestion
	Pulmonary oedema (acute case )
	pulmonary artery enlargement (if severe and long-standing)
	Calcified mitral annulus may be seen



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Widens carinal  
angle  $> 75^\circ$

LAA

LA

PV

May form right heart  
border

Posterior pushes the  
esophagus

Inferior (rare)

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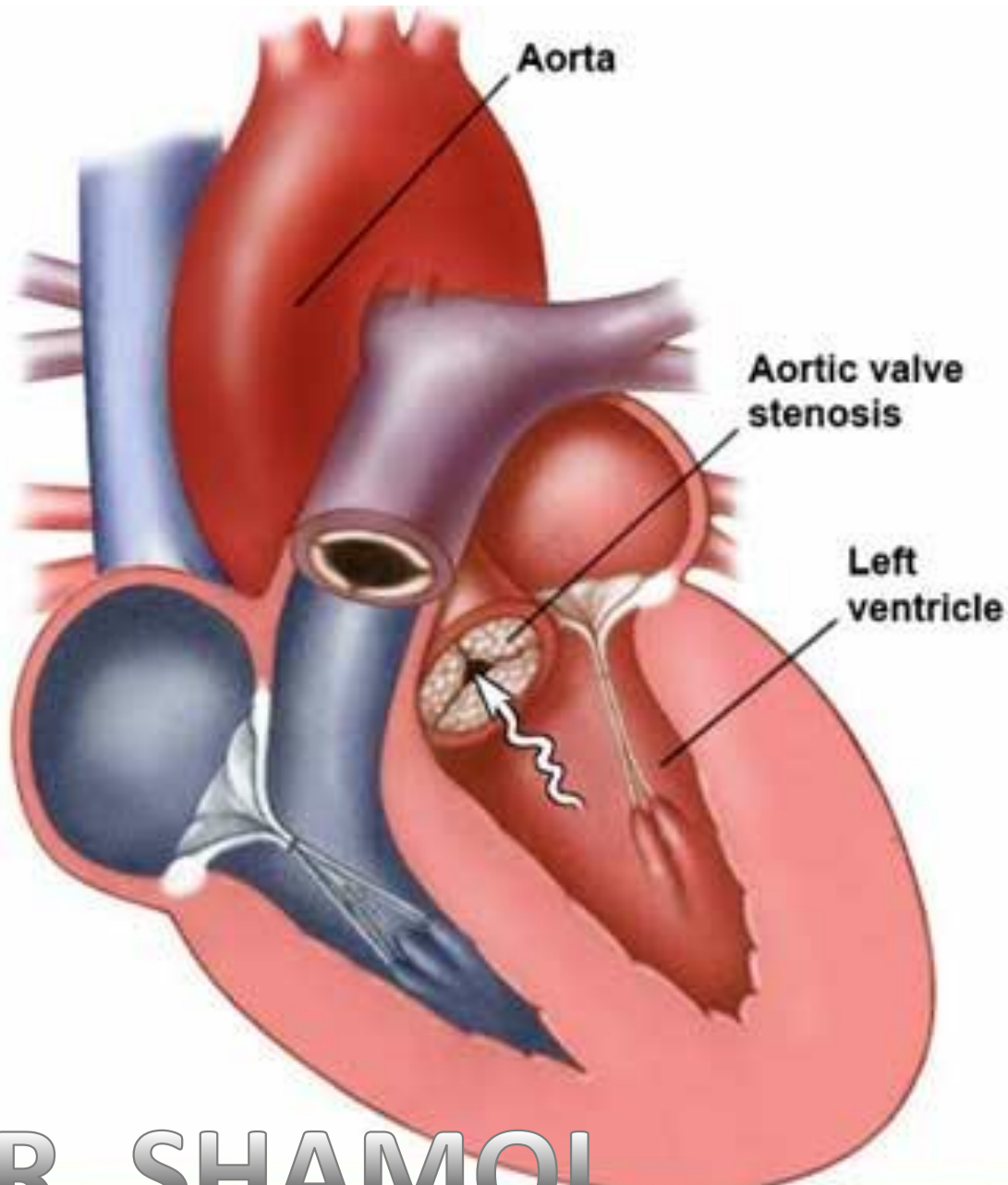
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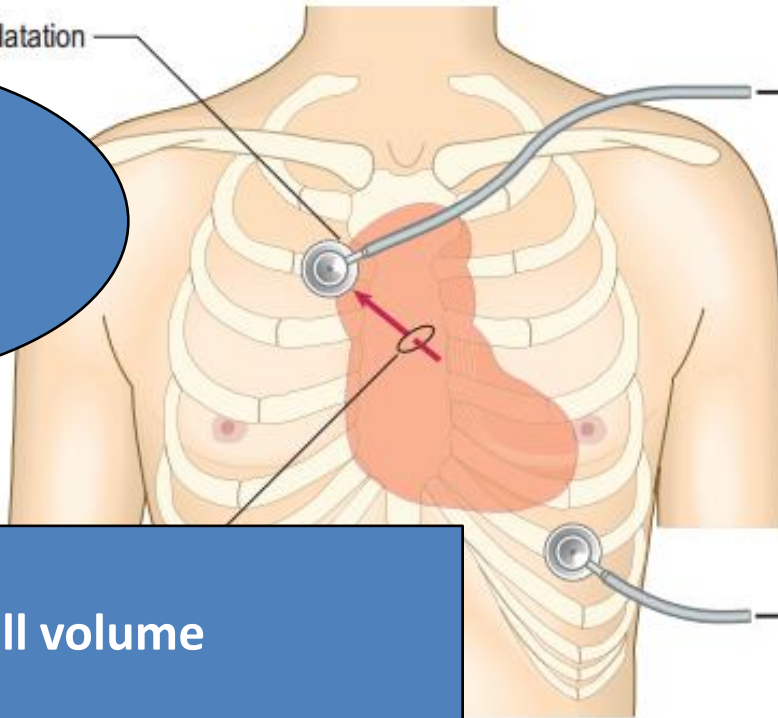
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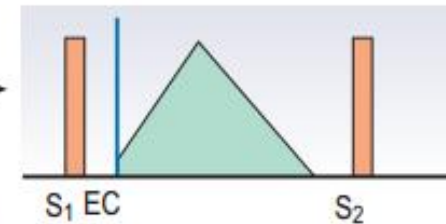
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## AORTIC STENOSIS

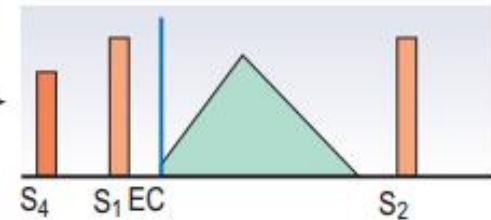
Aortic dilatation



The physical e



Lean patient forward with  
breath held in expiration to feel  
thrill and hear murmur best



**Pulse :**  
slow rising and small volume  
**BP:**  
low systolic & normal diastolic and  
narrow pulse pressure  
**apex beat :**  
heaving in nature and not shifted  
**thrill :**  
systolic thrill at aortic area .  
**Heart sound :**  
Soft second heart sound

## MURMUR

A harsh ejection systolic murmur in aortic area that best heard in sitting and leaning forward after breath hold after expiration.  
murmur radiates to right upper sterna edge, suprasternal notch radiates to the right neck over carotids

No medical treatments are proven to prevent or delay the disease  
Surgery is the treatment of choice

#### Indication for surgery

1. All symptomatic patient such as
  - a) syncope,
  - b) angina
  - c) symptoms of low cardiac output or heart failure
2. If mean systolic pressure gradient is  $> 50$  mm Hg (left ventricular systolic pressure  $>$  aorta)
3. If the valve area is  $< 0.7$  cm<sup>2</sup> (normal – 2.5 to 3 cm<sup>2</sup>)

#### type of surgical option

1. aortic valve replacement –the treatment of choice
2. Aortic balloon valvuloplasty
3. Valvotomy

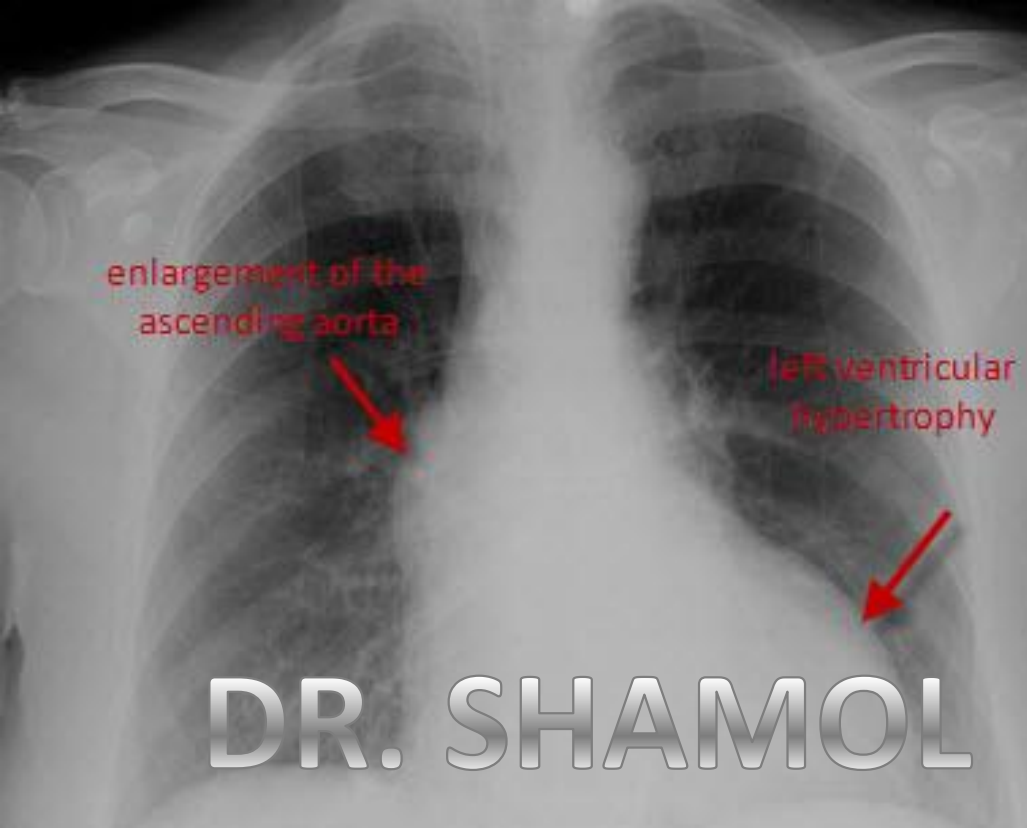
drug should be avoided

drugs that reduce after load e.g. GTN, ACE-I

In mild and asymptomatic case

- ❖ Use beta-blocker and
- ❖ periodic follow up for looking the symptoms like syncope
- ❖ Echocardiography

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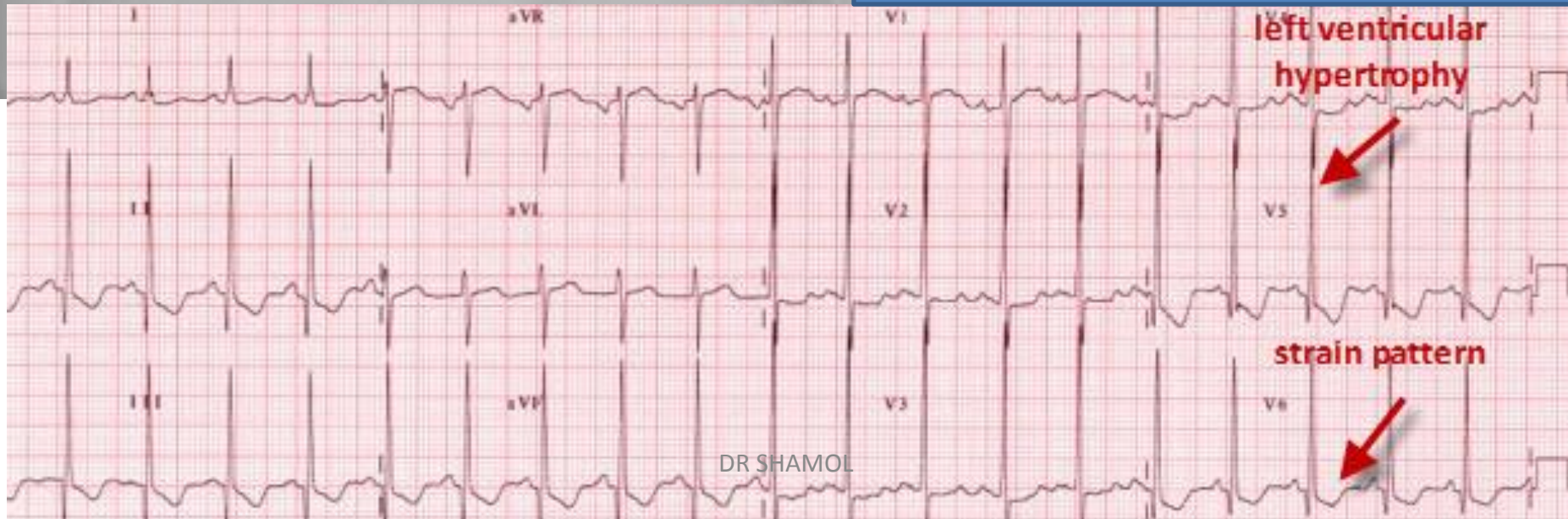
## ECG

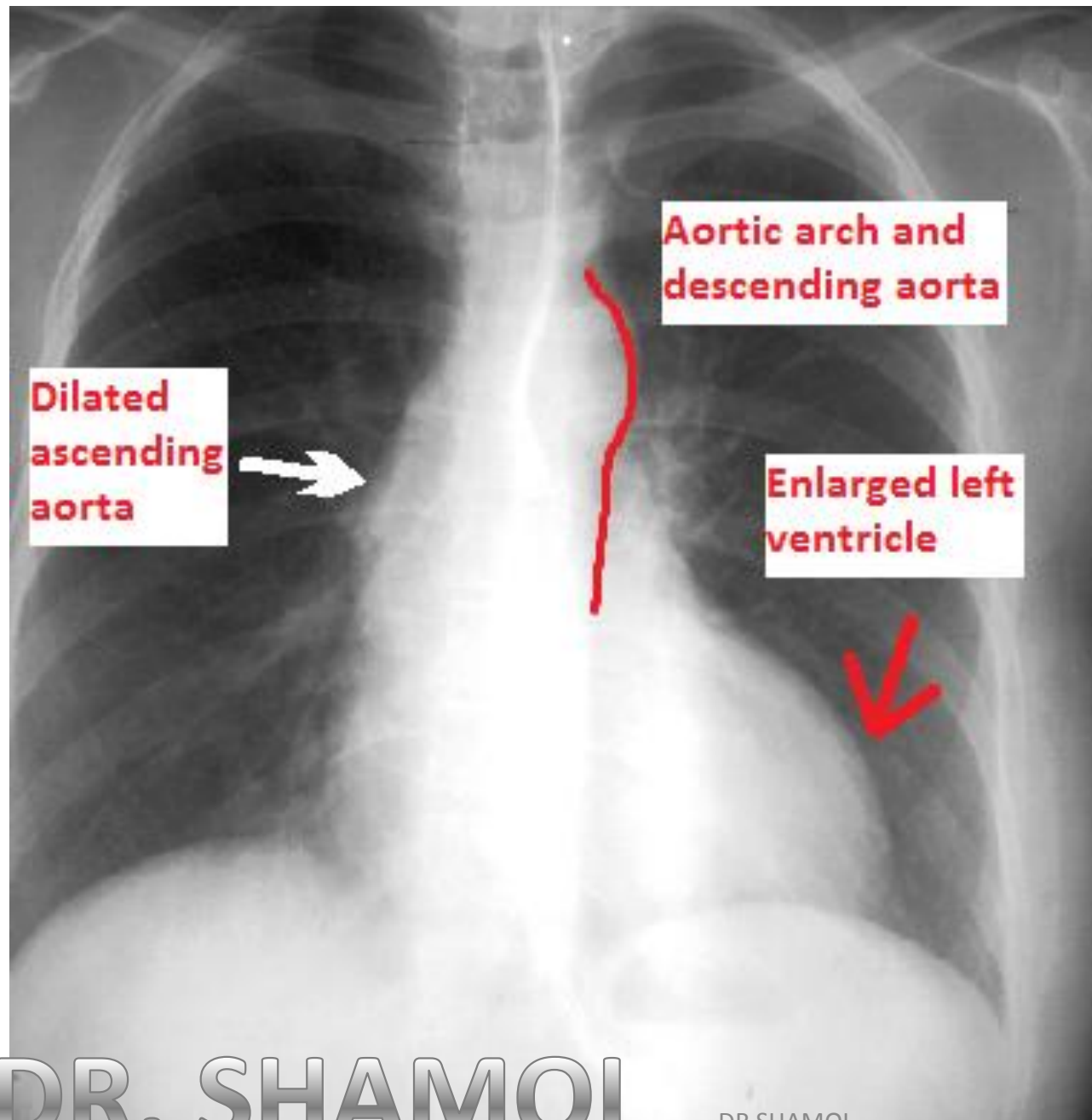
Left ventricular hypertrophy (usually) with down-sloping ST segments and T inversion ('strain pattern')

## Chest X-ray

May be normal

sometimes enlarged LV and dilated a  
Ascending aorta on PA view  
calcified valve on lateral view





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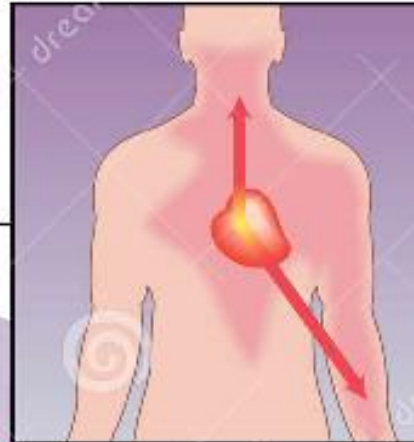


# Symptoms of Aortic Stenosis



Syncope  
(fainting)

Heart murmur,  
Angina pectoris



A	Asymptomatic-- usually Mild or moderate stenosis:
	Angina
B	Exertional Breathlessness
C	Exertional syncope
D	Sudden death
E	Episodes of acute pulmonary oedema



Air hunger  
(difficult breathing)



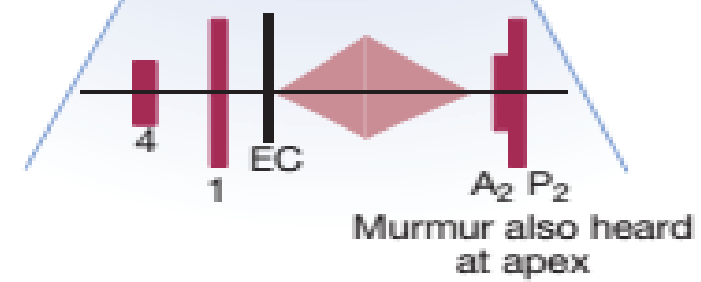
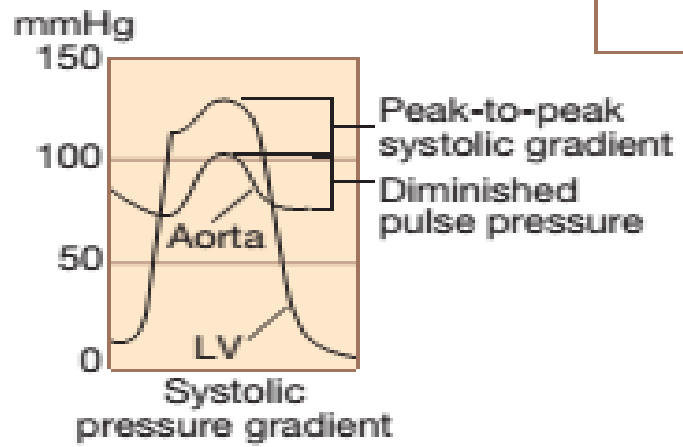
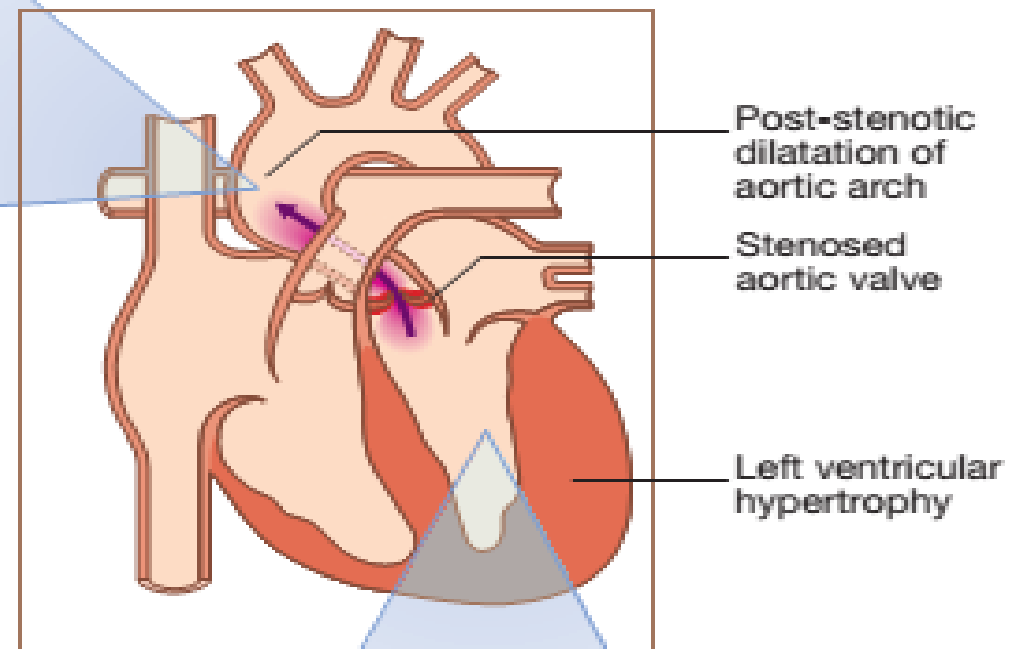
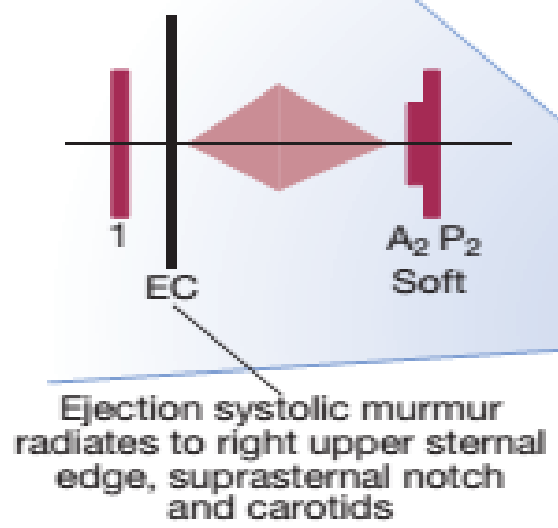
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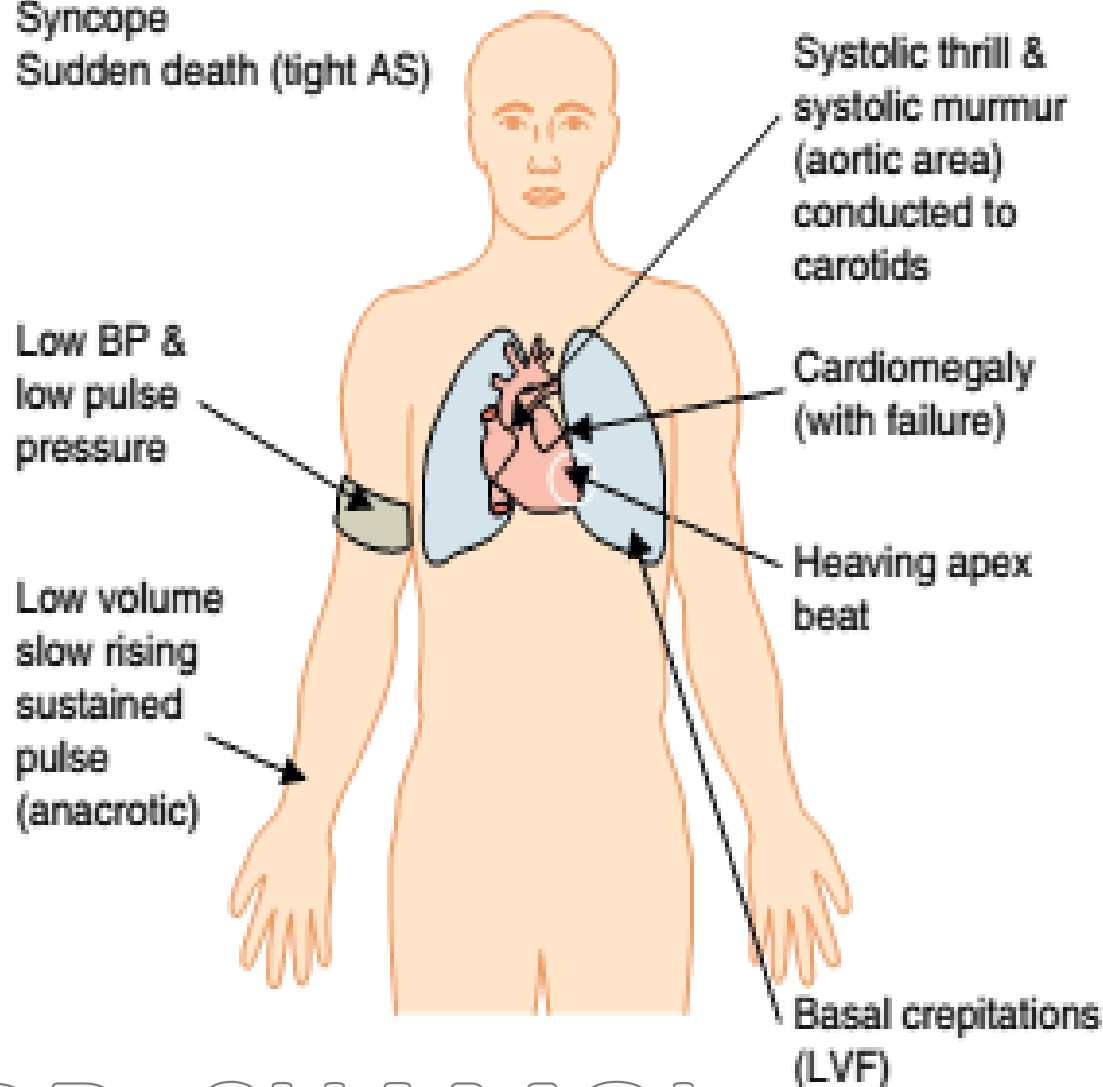
## Symptoms

Dyspnea

Angina pectoris

Syncope

Sudden death (tight AS)



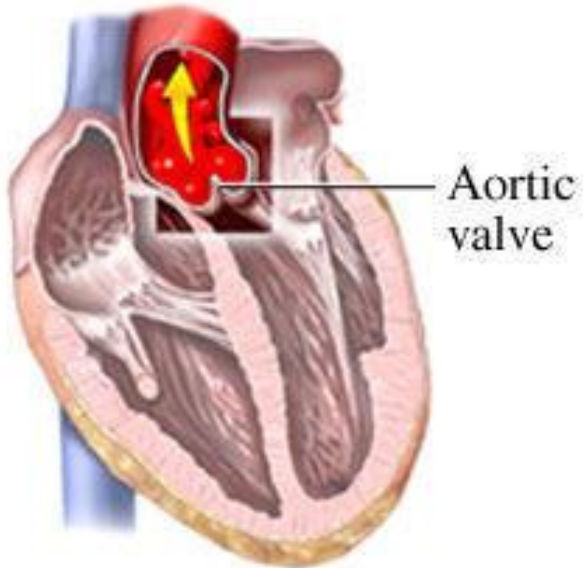
## Causes of aortic stenosis RBC

R--- Rheumatic aortic -- stenosis.

B--congenitally bicuspid aortic valve

C--  
calcification  
congenital

Normal valve operation



Valve closes after left ventricle pumps blood into aorta

Leakage of valve



Valve does not close completely, leaking blood into heart

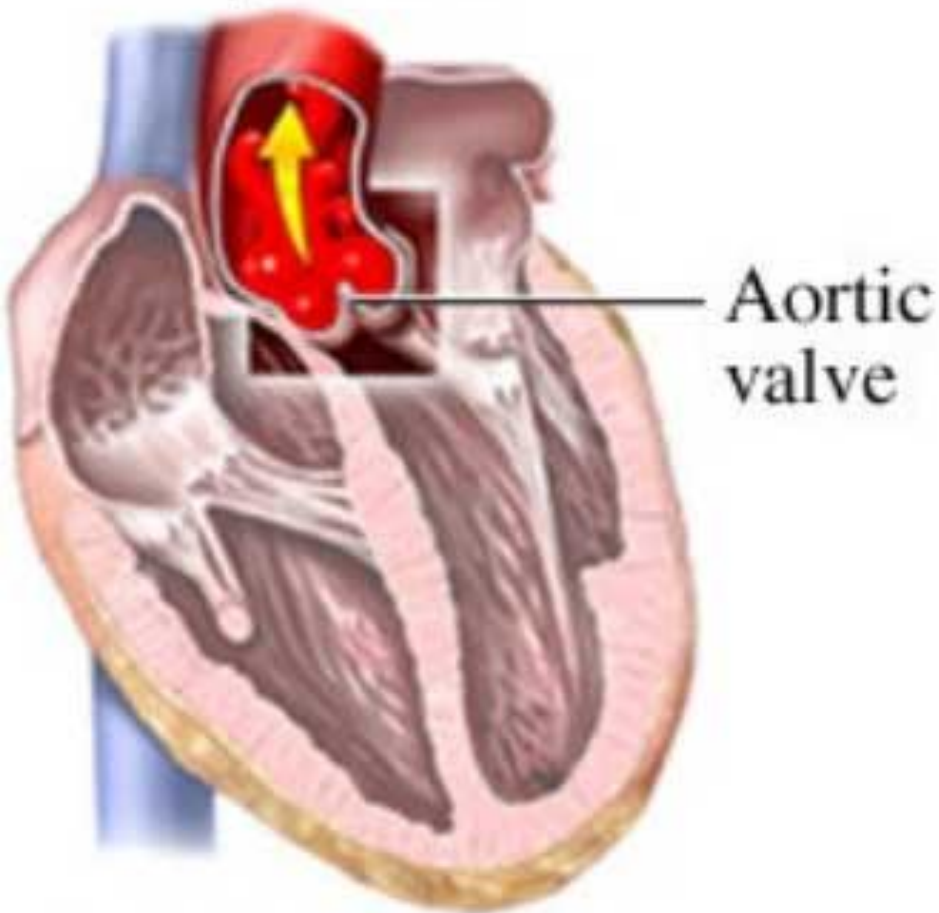


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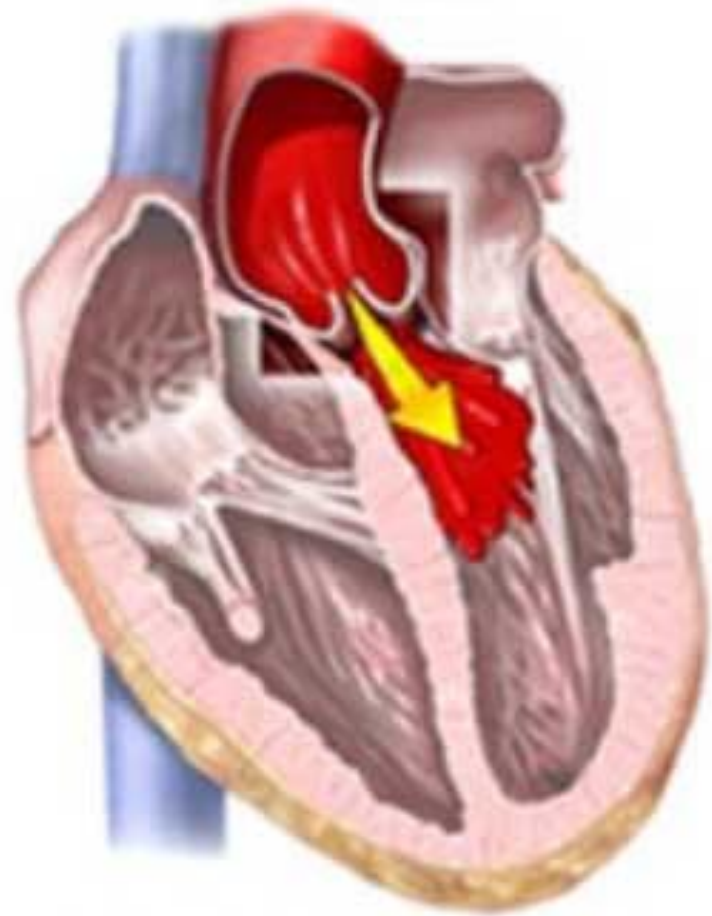


Normal valve operation



Valve closes after left ventricle pumps blood into aorta

Leakage of valve



Valve does not close completely, leaking blood into heart

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**pulse**

Collapsing pulse

**Dr**

high systolic, low diastolic,  
wide pulse pressure

Uncoiled  
aorta

Apex

Thrusting and shifted

**thrill**

Present in left parasternal area,  
diastolic in nature

**heart sound**

second heart sound A2—absent

Aortic valve

Lean patient forward with breath held



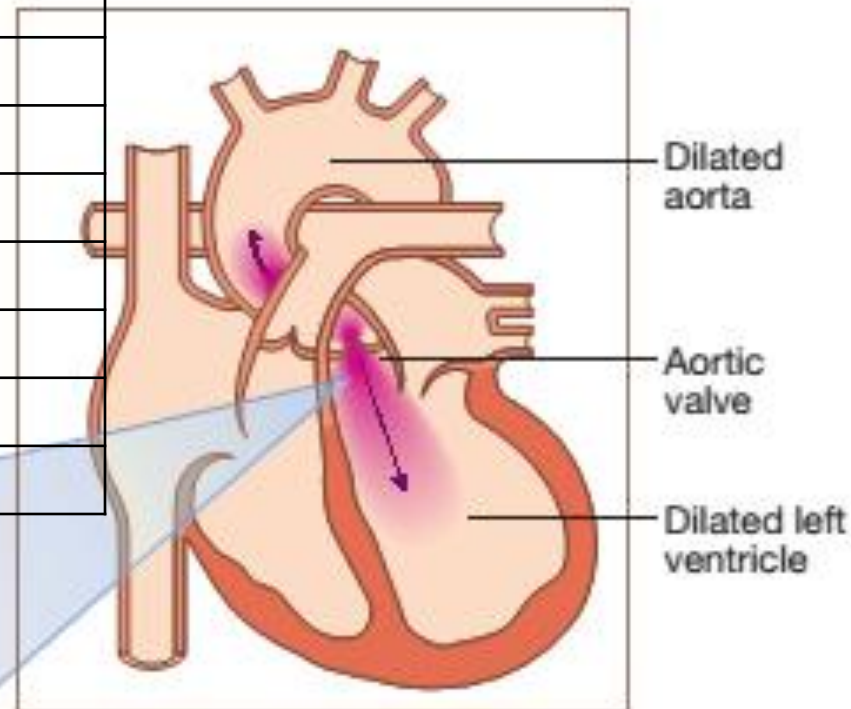
**murmur**

1. **early diastolic murmur**, which is high pitched, blowing, best heard in the left lower parasternal area with patient bending forward and breathing hold after expiration
2. A soft **mid-diastolic murmur** (Called Austin Flint murmur).
3. **Ejection systolic murmur** ---in aortic area which radiates to the neck, due to increased stroke volume and flow through the aortic valve

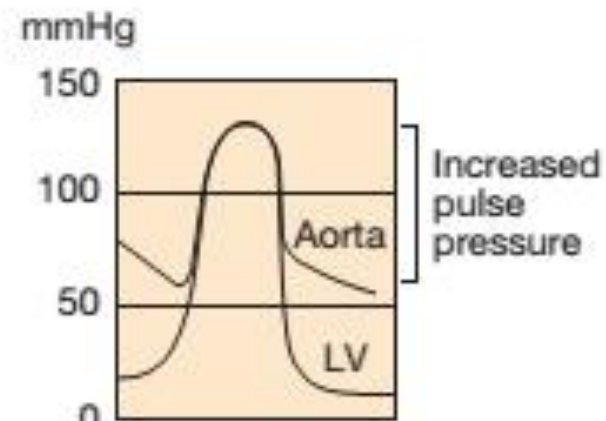
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Role of 3 in AR	3 pulse	Collapsing (water hammer)
		dancing carotid
		capillary pulsation
	3 BP	high systolic,
		low diastolic
		wide pulse pressure
	3 murmur	Early diastolic murmur
		Austin Flint murmur
		ejection systolic murmur



Lean patient forward with breath held in expiration to hear early diastolic murmur best



De Musset's sign  
(head nodding with  
each pulse)

Early diastolic  
murmur A1  
area radiating  
to Erb's area

Basal crackles  
(LVF)

Pistol shot sounds  
Duroziez murmur  
(femoral art)

Symptoms  
Palpitations  
Dyspnea  
Angina

Erb's area

LV  
enlargement

Hyperdynamic  
apex beat

Mid diastolic  
murmur mitral  
area (Austin  
Flint murmur)

Water  
hammer  
pulse

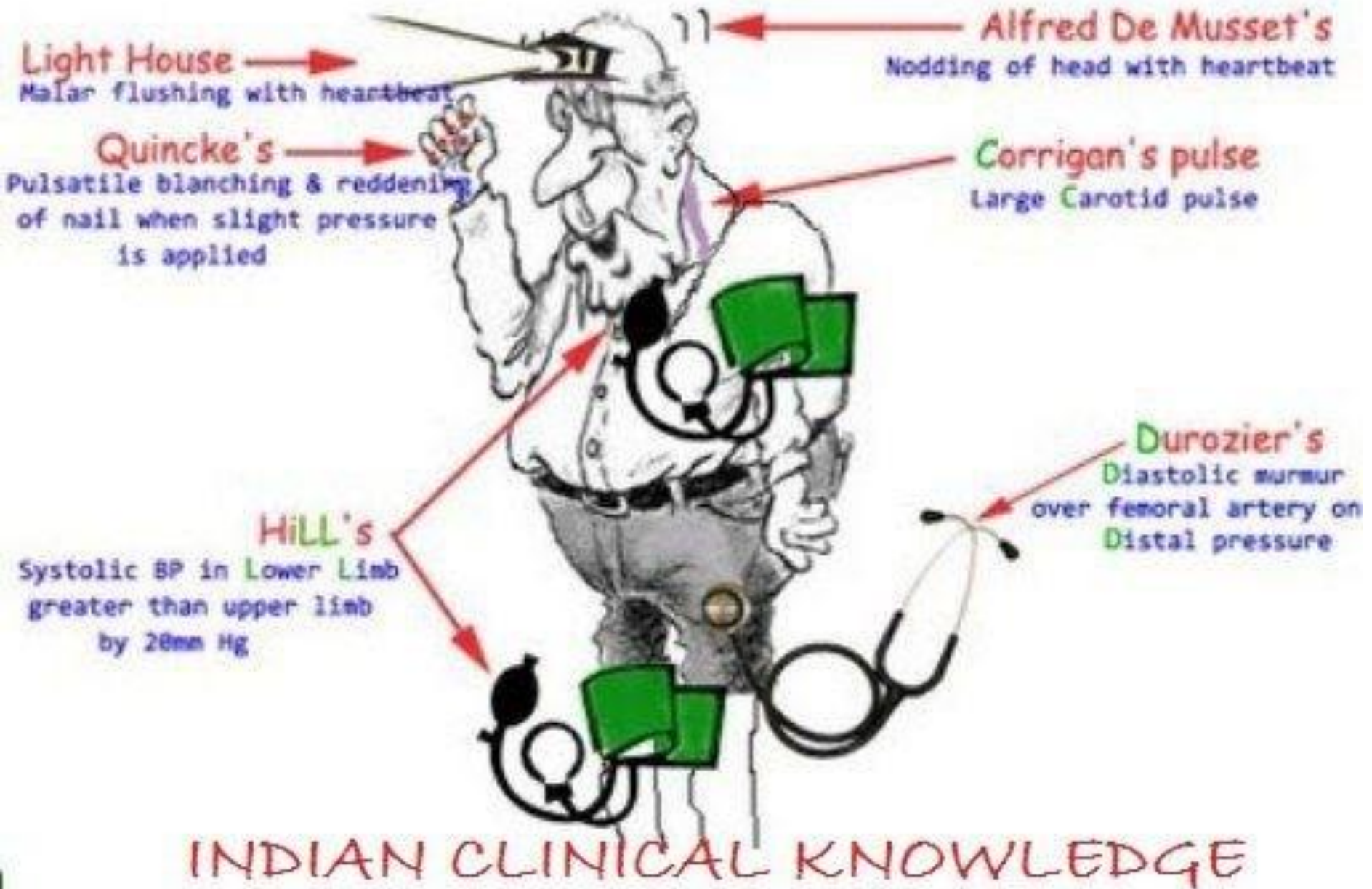
Hill's sign  
(BP in lower  
limbs > arms)

## peripheral sign in AR

D	Duroziez's sign or murmur	To and fro usually diastolic (may be systolic) murmur over the femoral artery on gradual compression of the vessel and ausculted proximally
	de Musset's sign	Head nodding with heart beat /each pulse
M	Mueller's sign:	Pulsation in uvula with heart beat
C	Collapsing pulse	large volume, rapid fall with low diastolic pressure
	Corrigan's sign	Visible carotid pulsation in neck or dancing carotids
	Capillary pulsation in figernails	Quincke's sign
H	Hill's sign:	High BP in legs than arms (high systolic BP, 60 mm difference)
Pistol	'pistol-shot' Femoral bruit or Traube's sign	A booming sound heard over femorals



type of treatment	medical		
	surgical		
indications of surgery:	All Symptomatic patient		
	Asymptomatic patient 50—55--50	LV systolic dysfunction (EF < 50%)	
		LV dilatation	LV end systolic dimension >55 mm
			LV end diastolic dimension >75 mm
		Aortic root dilatation >50 mm	
Medical management	Asymptomatic	routine follow-up with ECHO	
	where surgery contraindicated	Vasodilators (ACE-Is, calcium-channel blockers) offer good symptomatic relief and may improve haemodynamic profile	
		Rx	LV dysfunction → Digoxin, ACE inhibitor, diuretic
			Heart failure → Digoxin, ACE inhibitor, diuretic
		Anginal pain	treated with nitrates but use B-blockers with caution
		systolic BP	should be controlled with vasodilating drugs, such as nifedipine or ACE inhibitor



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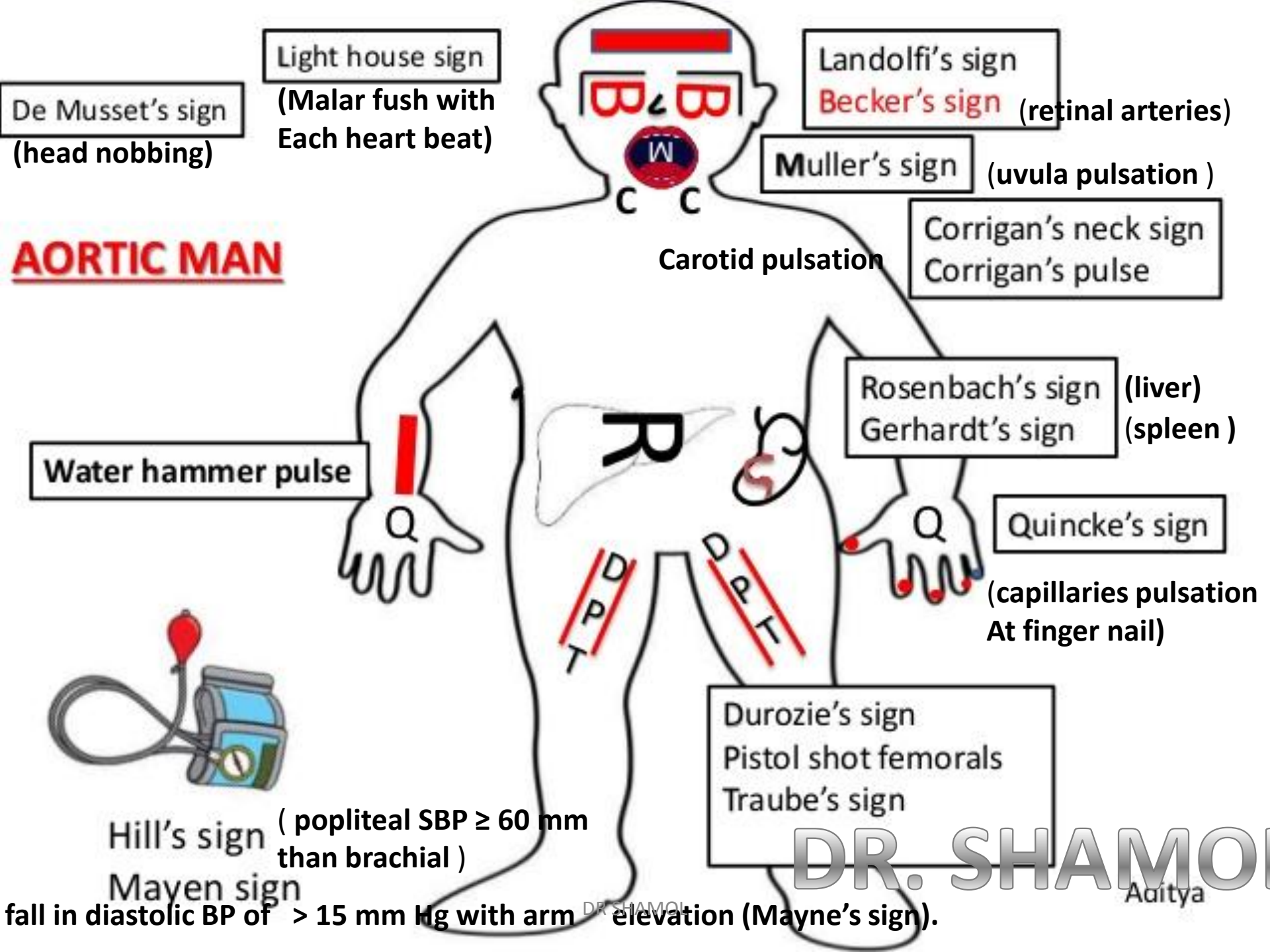
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# Congenital Bicuspid valve or disproportionate cusps

involvement of valve: RIB-T	R- Rheumatic fever	
	I-- Infective endocarditis	
	B- Bicuspid aortic valve	
	T- Trauma	
Aortic root dilatation:	myxomatous degeneration	Marfan syndrome
		EhlersDanlos syndrome
	disease of aorta	Hypertension
		aortic aneurysm
		aortic dissection
		Rupture of sinus of Valsalva
		supravalvular aortic stenosis
	arthritis	Seronegative arthritis (ankylosing spondylitis, Reiter's syndrome)
		SLE & Rheumatoid arthritis
	Osteogenesis imperfect	
	infection	Syphilitic aortitis
	arteritis	(Takayasu's, giant cell).

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De Musset's sign  
(head nobbing)

Light house sign  
(Malar flush with  
Each heart beat)

Landolfi's sign  
Becker's sign (retinal arteries)

Muller's sign (uvula pulsation )

Corrigan's neck sign  
Corrigan's pulse

Carotid pulsation

Rosenbach's sign (liver)  
Gerhardt's sign (spleen )

Quincke's sign

(capillaries pulsation  
At finger nail)

Water hammer pulse



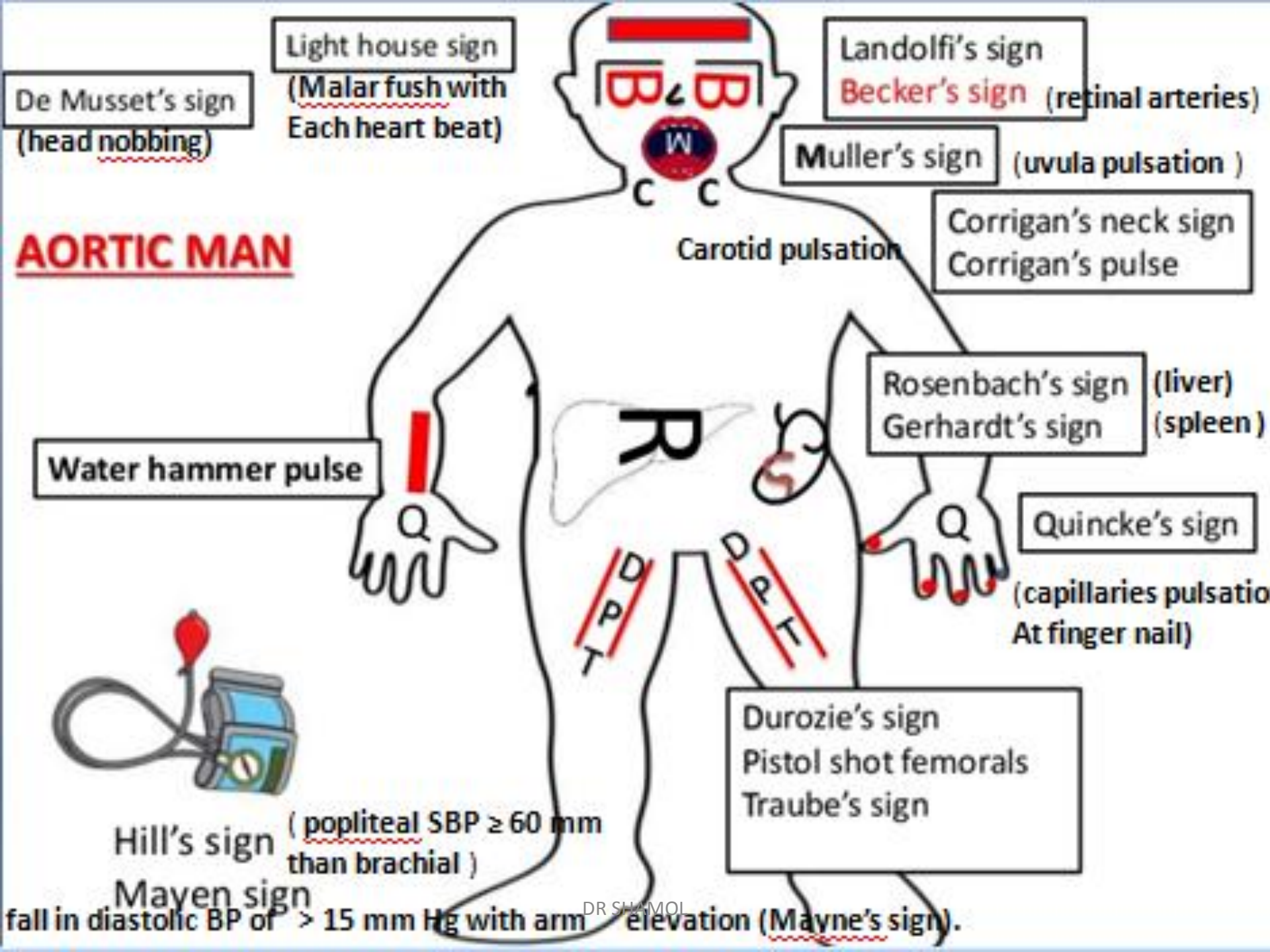
Hill's sign ( popliteal SBP ≥ 60 mm  
than brachial )

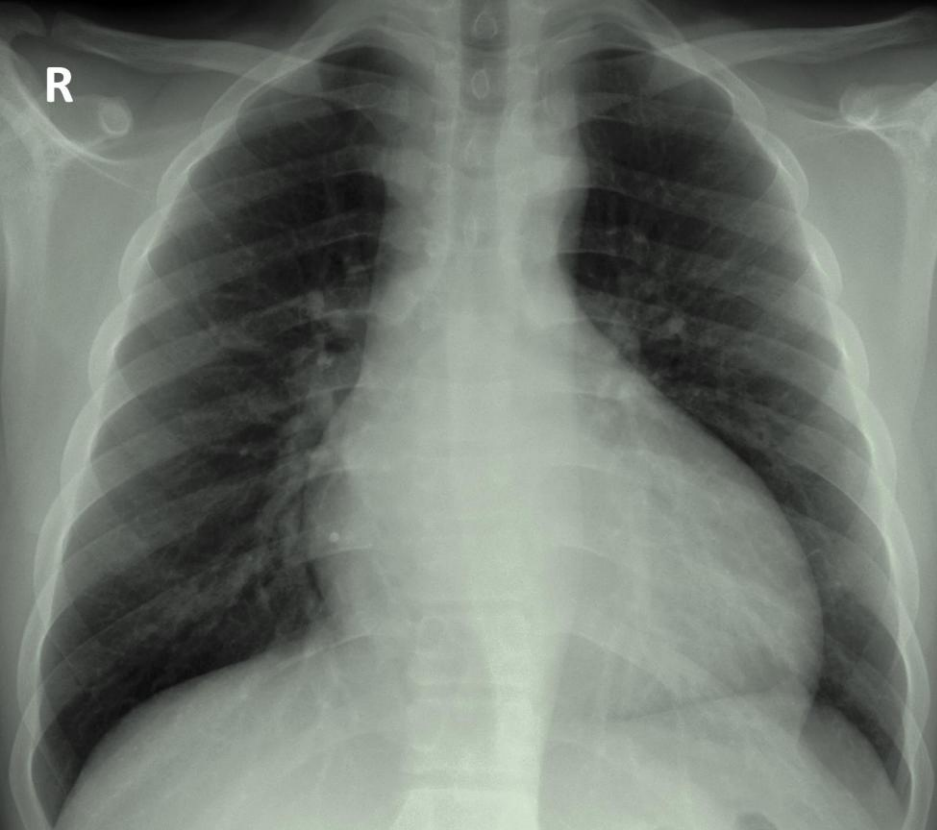
Mayen sign

Durozie's sign  
Pistol shot femorals  
Traube's sign

fall in diastolic BP of > 15 mm Hg with arm elevation (Mayne's sign).



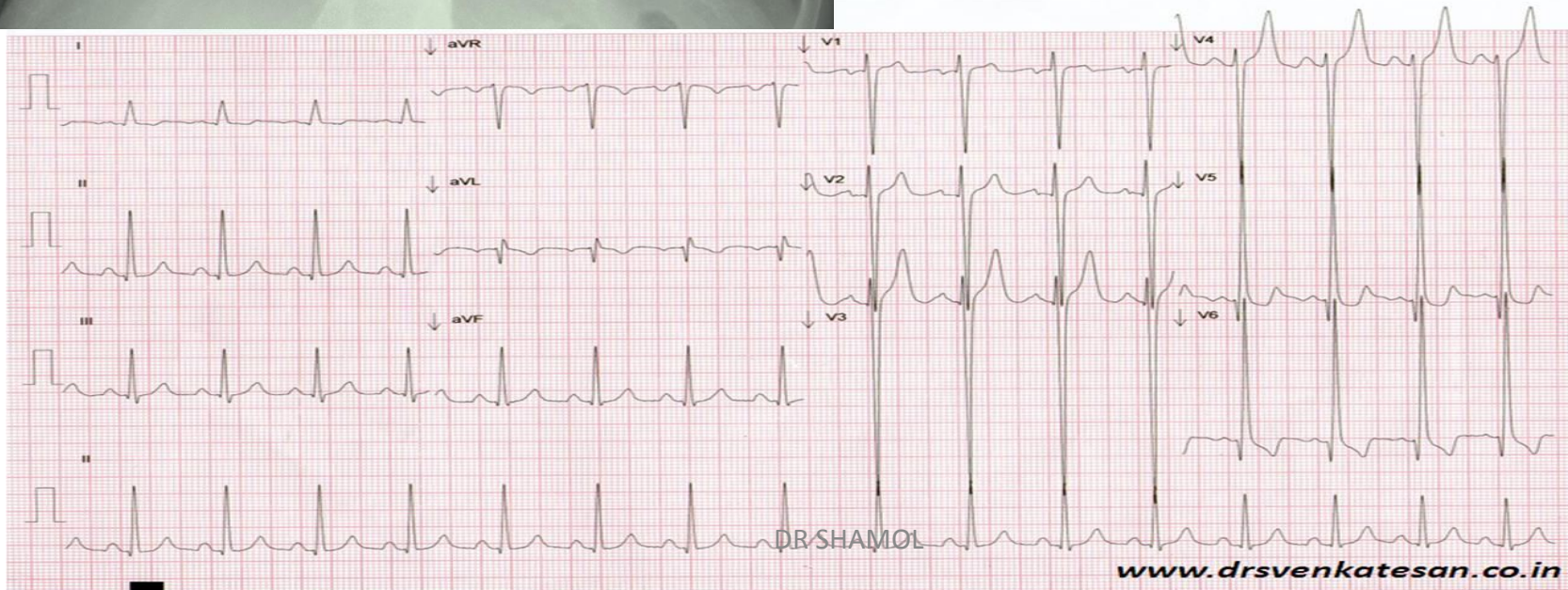




ECG	Initially normal,
	later left ventricular hypertrophy and T-wave inversion
Chest X-ray	Cardiomegaly
	Features of left heart failure

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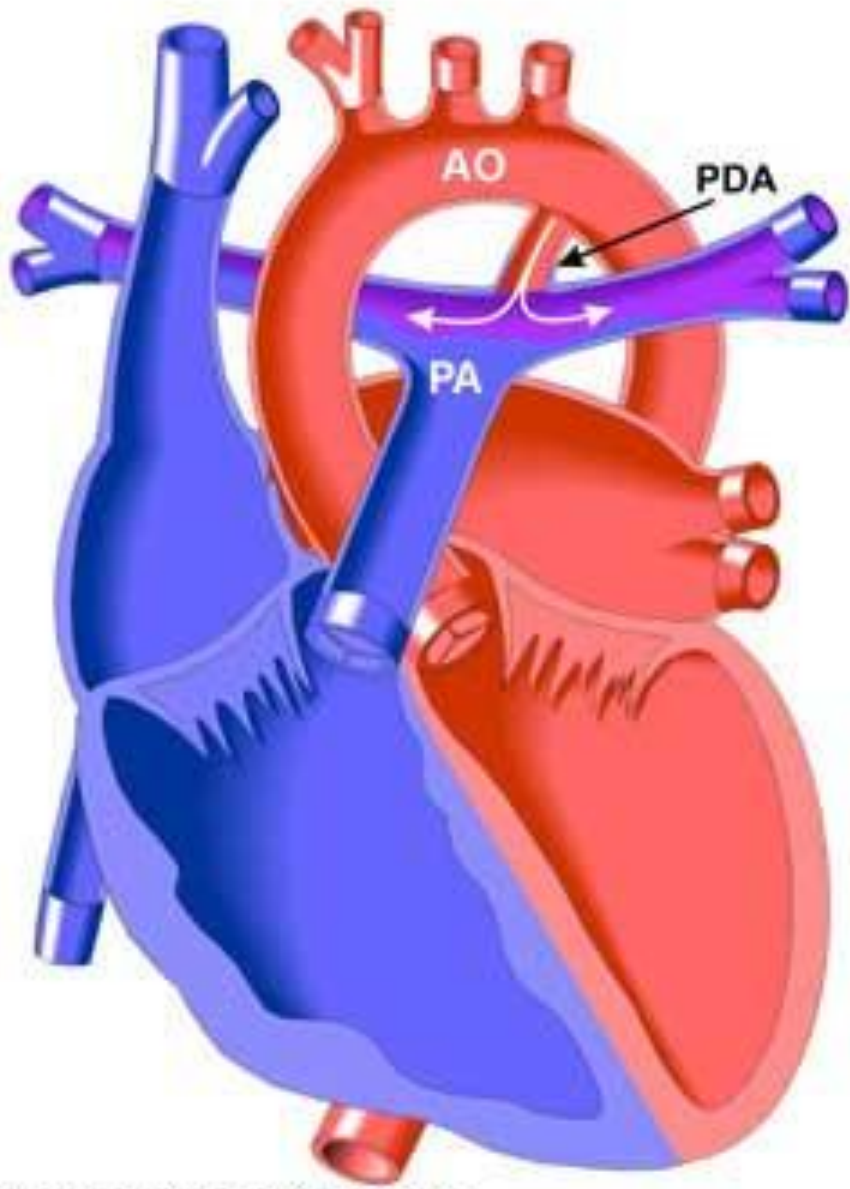
**V 5 , V6 in volume**



<b>Symptoms</b>	A	Asymptomatic--
		Awareness of heart beat, 'palpitations'
	B	Breathlessness
	C	chest pain or angina
	D	feature of underlying diseases
	E	Episodes of acute pulmonary oedema
	F	acute heart failure or cardiogenic shock

acute AR	D→	Dissecting aneurysm affecting ascending aorta
	A→	Acute rheumatic fever (due to valvulitis)
	T→	Trauma.
	A→	Acute bacterial endocarditis





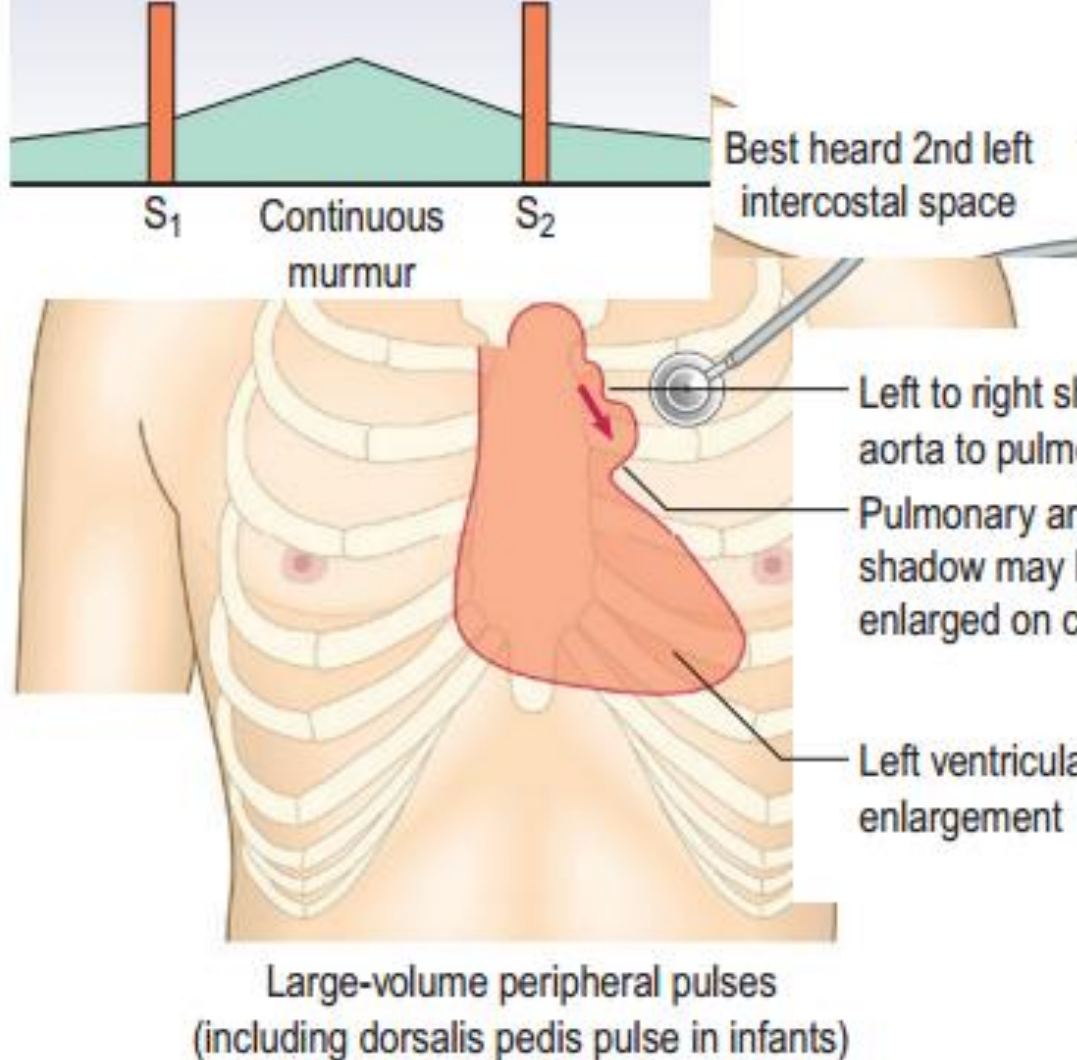
**Patent Ductus Arteriosus**

P D A

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Pulse : Collapsing pulse

if reverse  
shunt

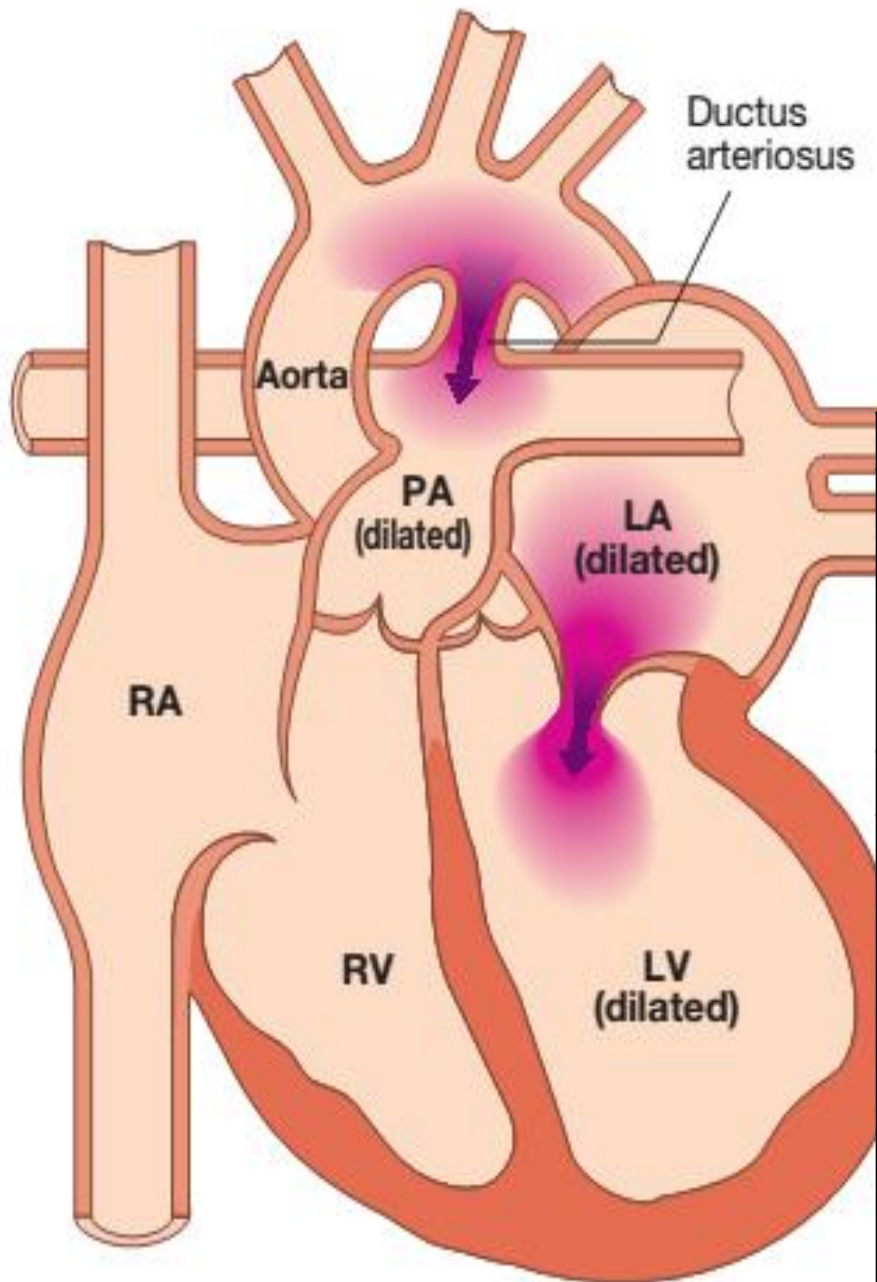
Cyanosis in lower limb --  
differential cyanosis--  
clubbing in lower limb--  
differential clubbing

apex beat : heaving and  
shifted

thrill:  
Systolic and/or  
diastolic thrill in the  
left second intercostal  
space

**murmur** : Loud, continuous 'machinery' murmur in left 2nd and 3rd intercostal space below the clavicle radiates to the neck Heaving (murmur is prominent on expiration, may be heard posteriorly)

The murmur begins after the first heart sound, peaks with the second sound, and trails off in diastole—known as Gibson murmur



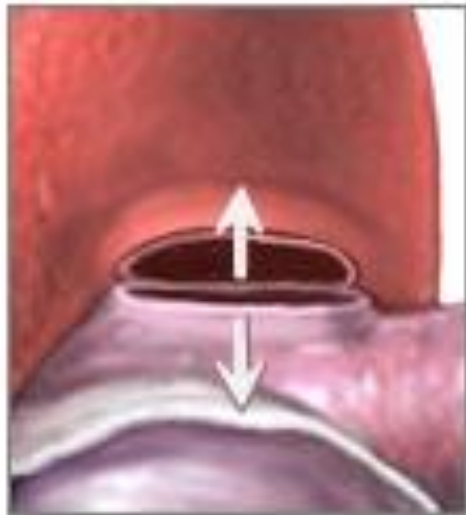
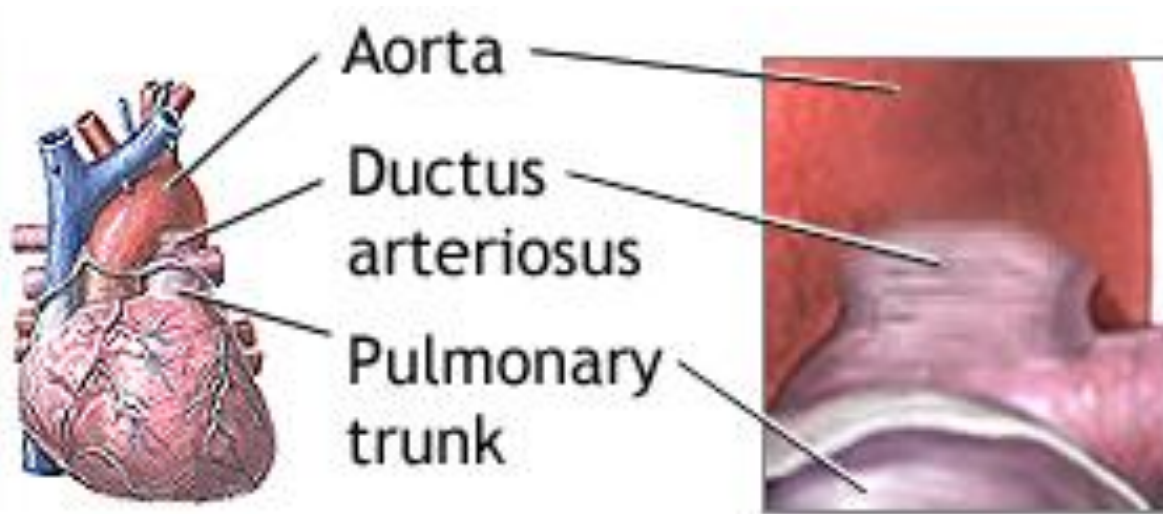
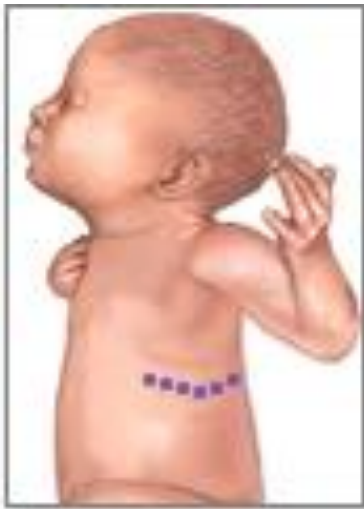
**Persistent ductus arteriosus:**  
There is a connection between the aorta and the pulmonary artery with left-to-right shunting.

#### Differential diagnoses

- ❖ Arteriovenous fistula (coronary, pulmonary or systemic)
- ❖ Venous hum
- ❖ Rupture of sinus of Valsalva to the right ventricle or atrium

#### name some causes of continuous murmur

- ❖ PDA
- ❖ Arteriovenous fistula (coronary, pulmonary or systemic)
- ❖ Venous hum
- ❖ Rupture of sinus of Valsalva to the right ventricle or atrium
- ❖ Mitral regurgitation murmur with aortic regurgitant murmur
- ❖ Ventricular septal defect with aortic regurgitation

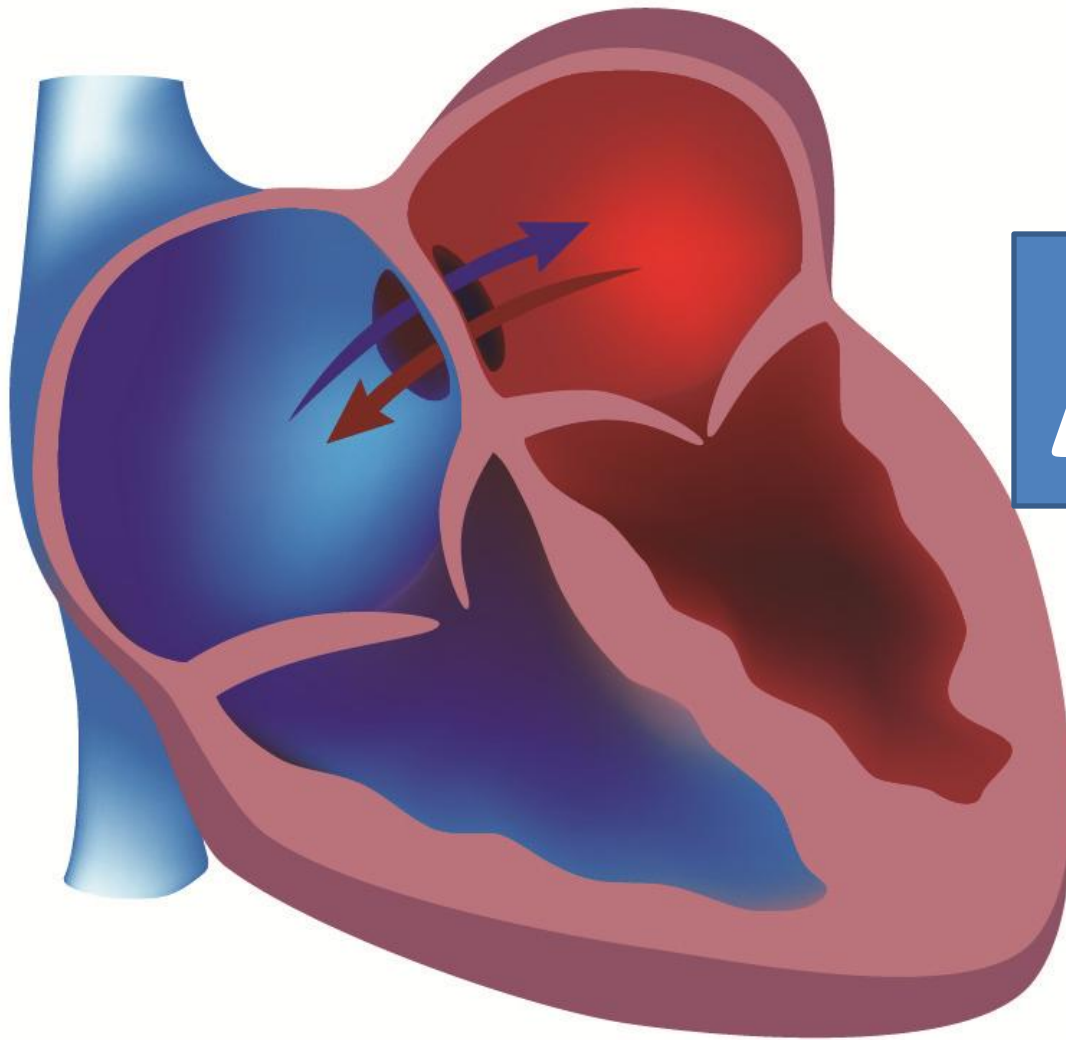


The aorta and pulmonary trunk are separated



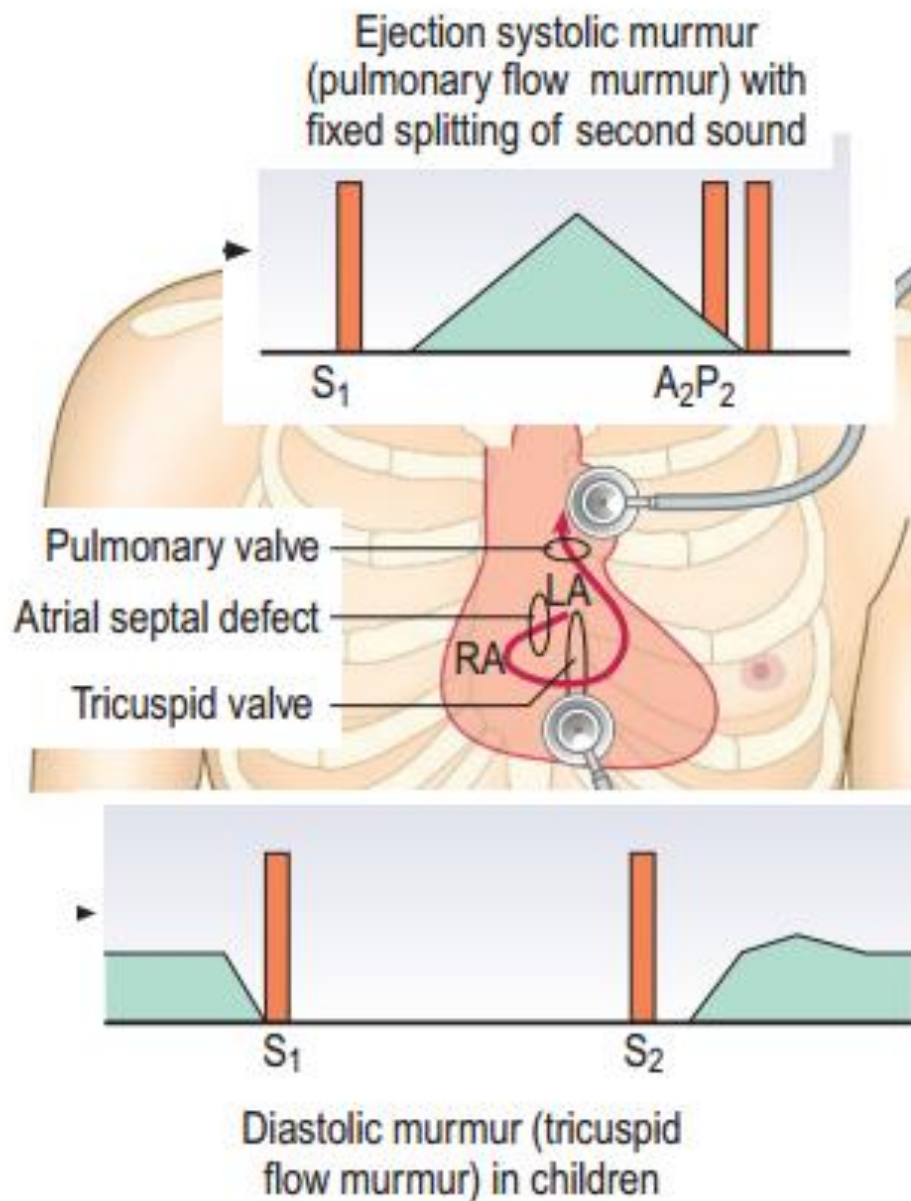
The open ends are closed

# *Atrial Septal Defect*



A S D





wide, fixed splitting of the second heart sound:

Ejection systolic flow murmur in the left second and third intercostal space (due to increase flow to pulmonary valve )

In case of large shunt, there may be a diastolic flow murmur over the tricuspid valve.

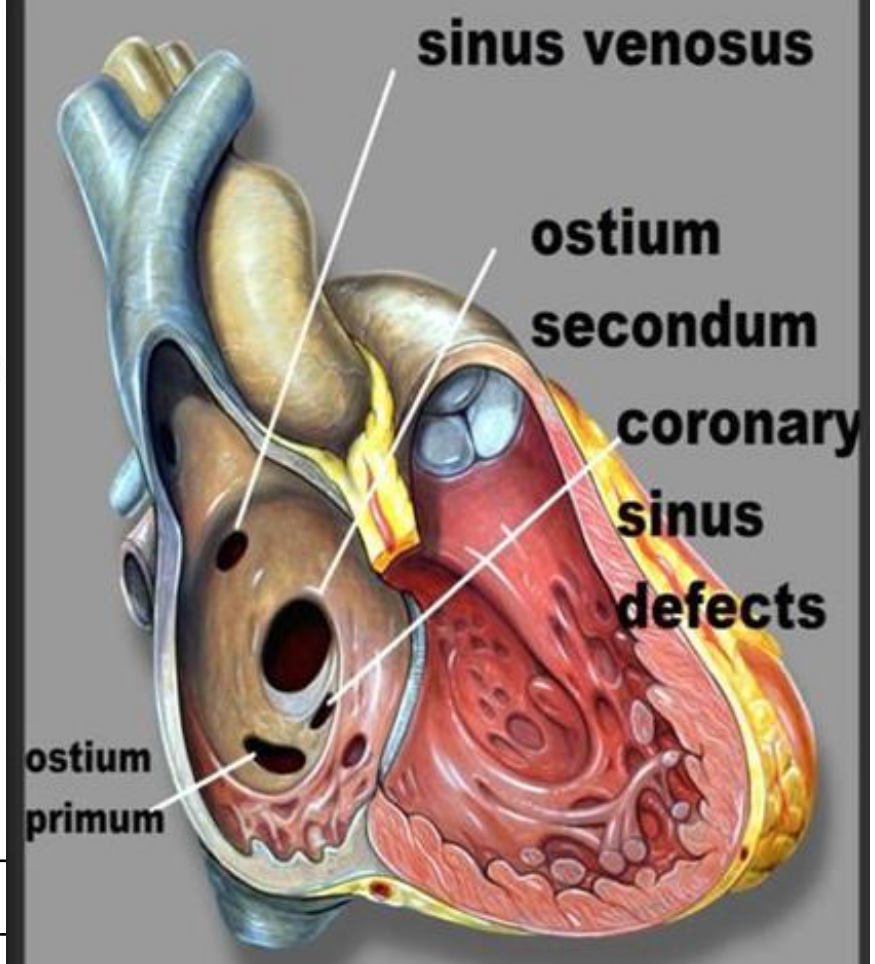
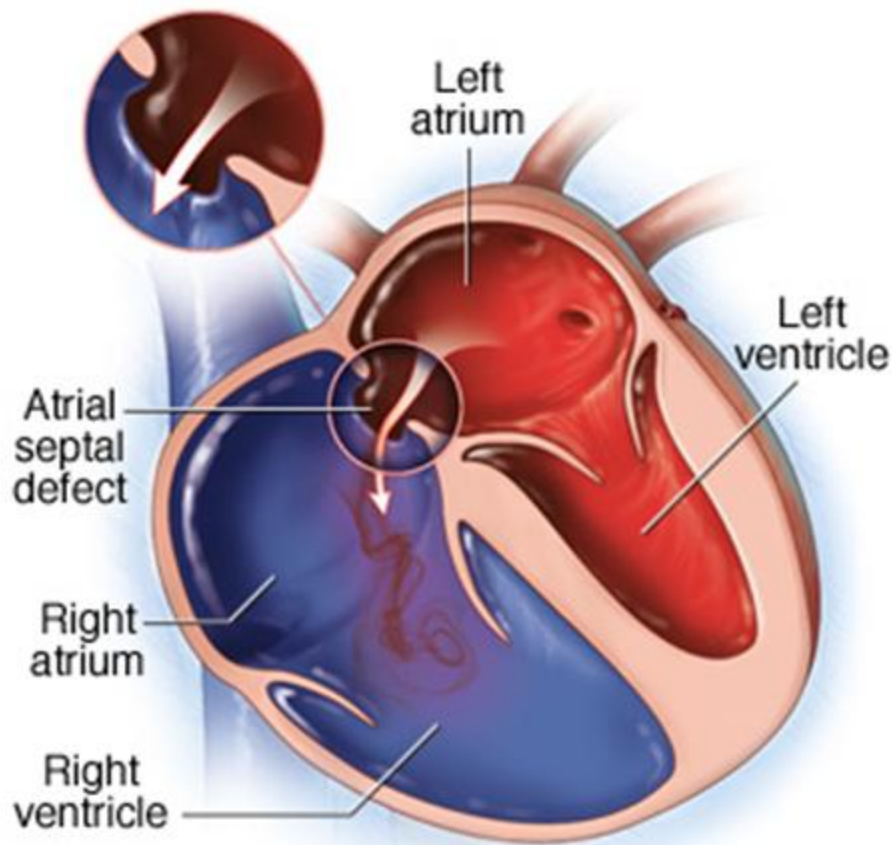
Left parasternal heave

If patient developed pulmonary hypertension then you find following

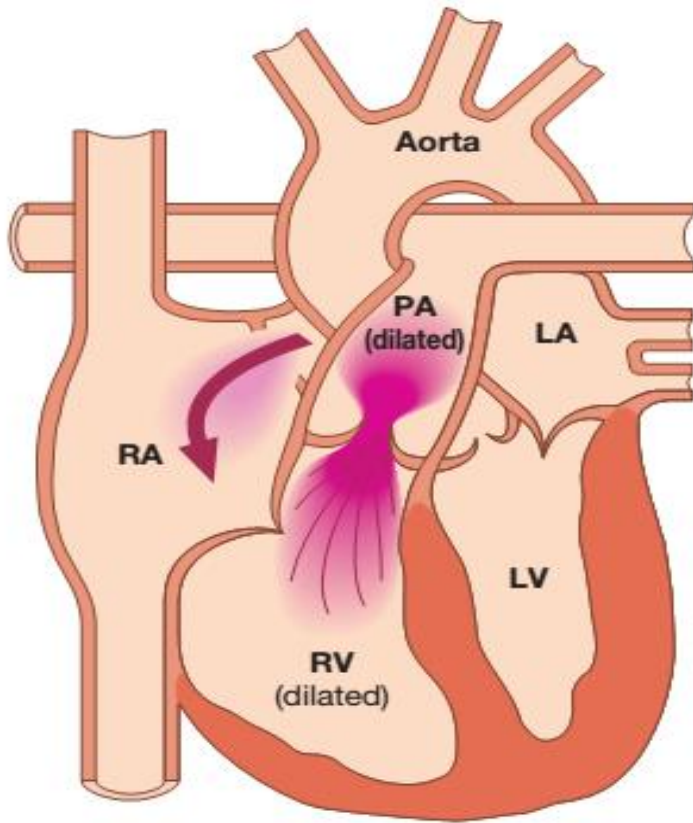
- ❖ palpable P2
- ❖ Left para-sternal heave

Auscultation

- ❖ loud P2



A	arrhythmias--AF
B	breathlessness
C	chest infection
D	detected at routine clinical examination or following a chest X-ray
E	paradoxical emboli
F	heart failure



## Atrial septal defect.

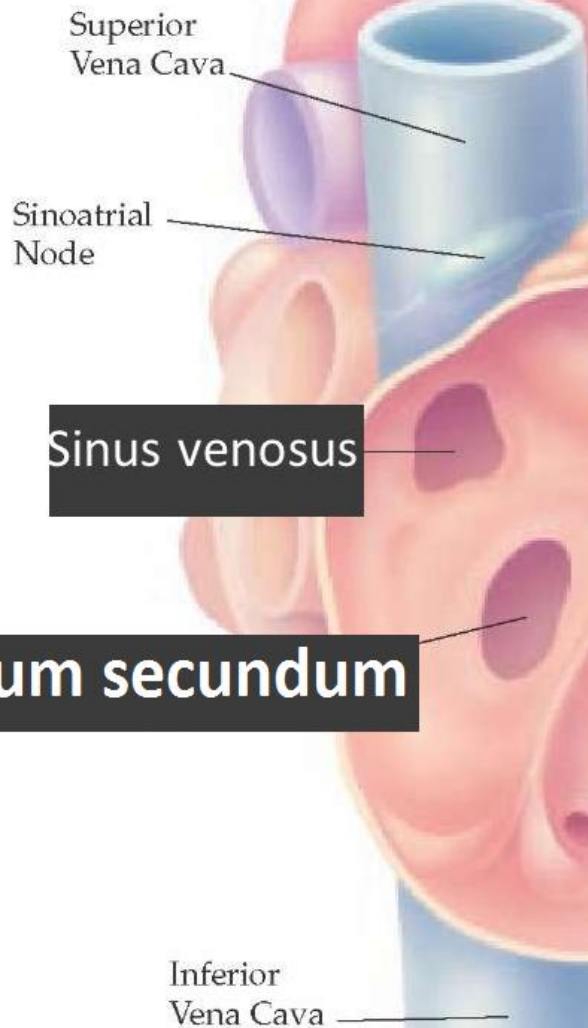
Blood flows across the atrial septum (Arrow) from left to right.

The murmur is produced by increased flow velocity across the pulmonary valve, as a result of left-to-right shunting and a large stroke volume.

complication of <b>CAPSIRE</b>
C—chest infection
A—arrhythmia
P—PTH
S—stroke
I—infective endocarditis
R—Right vent failure
E—Eisenmenger



# TYPE OF ASD



Ostium secundum	The defect involving the middle portion of the atrial septum named the fossa ovalis/ foramen ovale
Ostium primum	defect in the lower part of the septum
Sinus venosus type	defect in the septum just below the entrance of the superior vena cava into the right atrium

**ostium secundum**

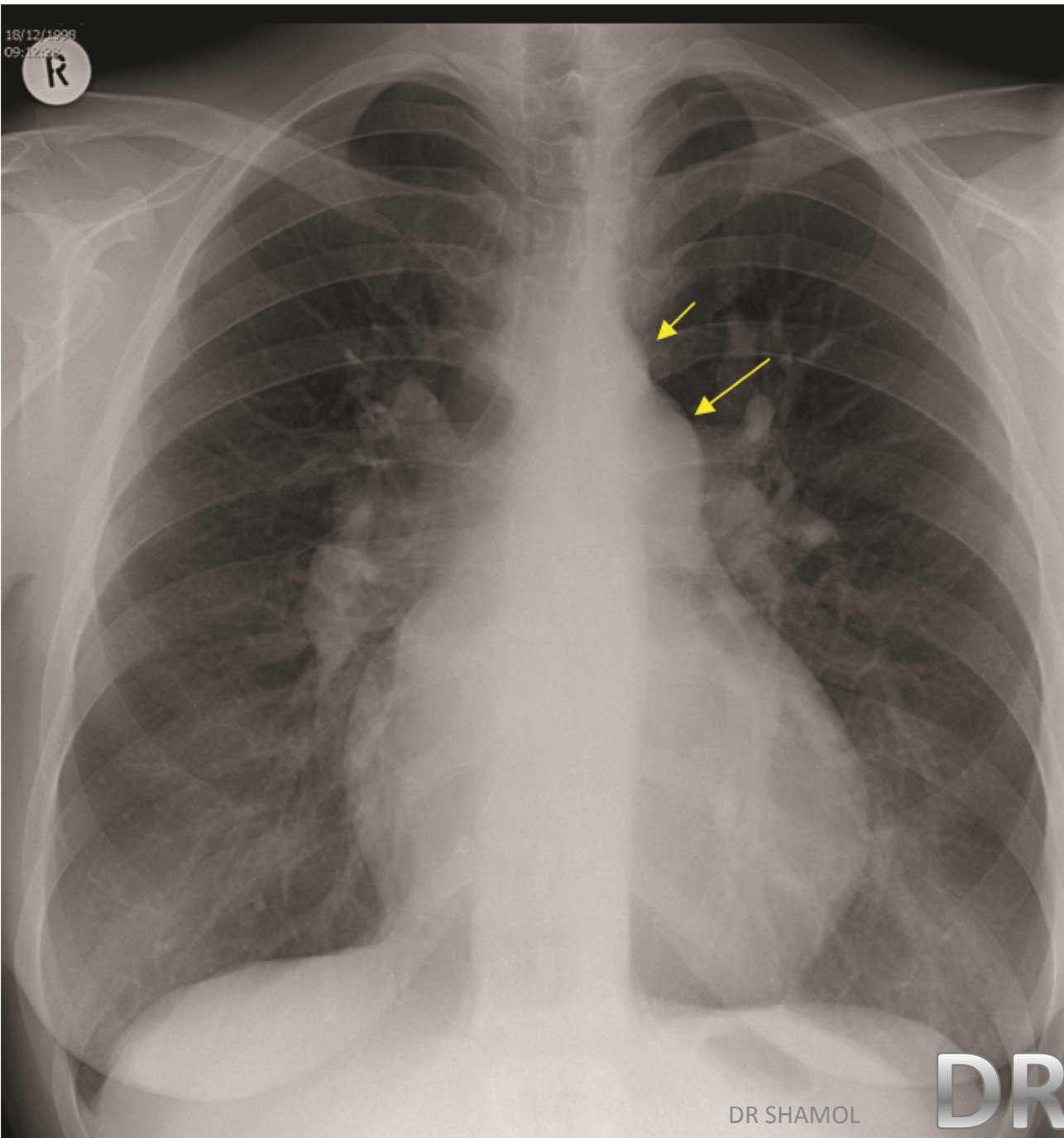
**ostium primum**



treatment of choice	surgical closure	
in case child	If detected in early childhood surgical closure is recommended	
	usually done between the age of 5 and 10 years	
in case of adult	Small ASD	Surgery is not needed,
		only follow-up should be done
		the patient usually lives a normal life
	Moderate to large	Surgical closure should be done if pulmonary flow is increased 50% above systemic flow (pulmonary flow to systemic flow is 1.5:1)
	surgery done	Closure can be done by percutaneous cardiac catheterisation using implantable closure devices (clamshell devices)
	prognosis	Those operated on before the age of 25 years have an excellent prognosis
contraindication to surgery	developed Eisenmenger's syndrome	
	severe pulmonary hypertension	

18/12/1998  
09:12:23

R

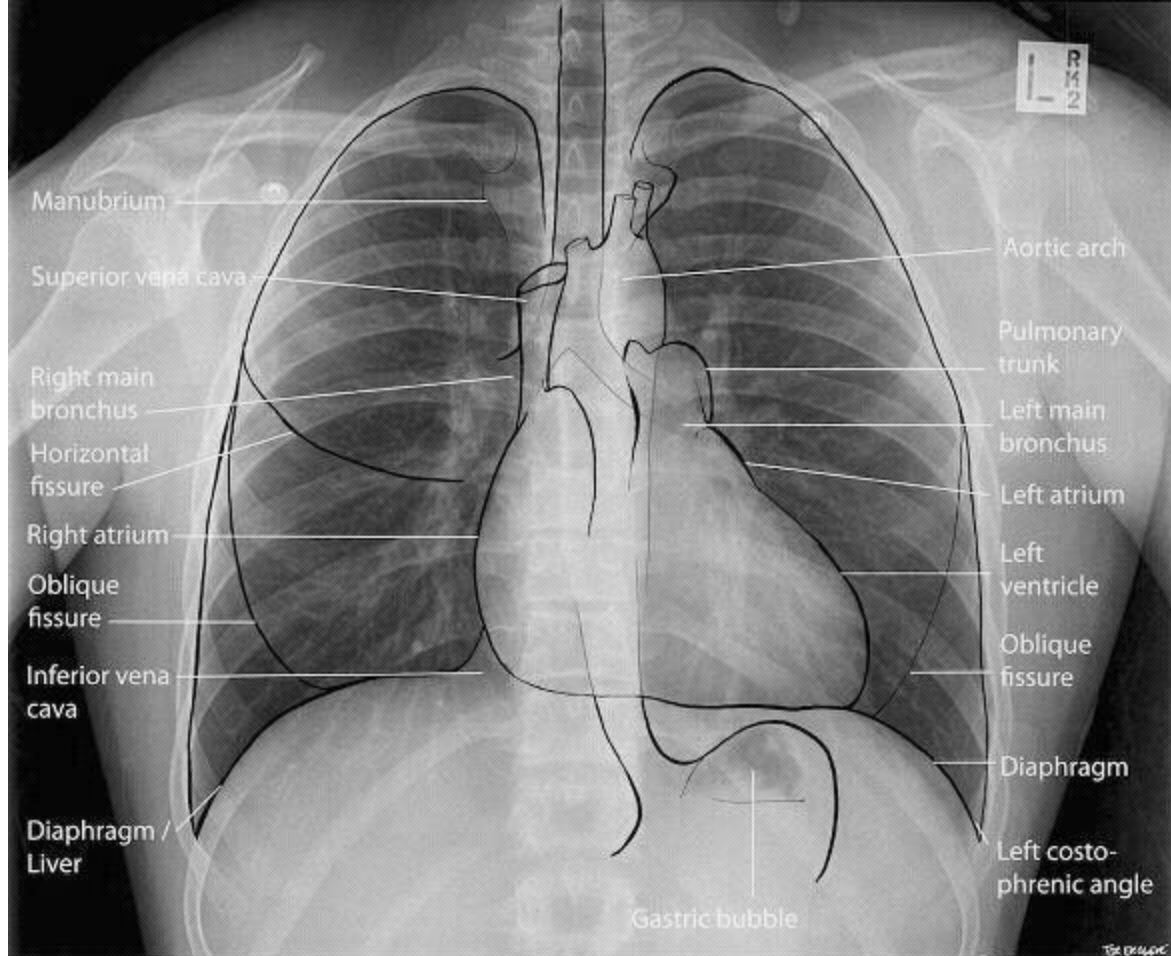


large pulmonary conus  
(Prominent pulmonary  
arteries)

Small aortic knob

**cardiomegaly** RV type /  
enlargement of the heart  
(Enlarged RV and right  
atrium)

plethoric lung field /  
pulmonary plethora /A  
peripheral pulmonary  
vascular pattern of 'shunt  
vascularity' (in which the  
small pulmonary arteries are  
especially well visualized in  
the periphery of both lungs)



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18/12/1998  
09:12:23

R

Small aortic knuckle

Pulmonary clonus full

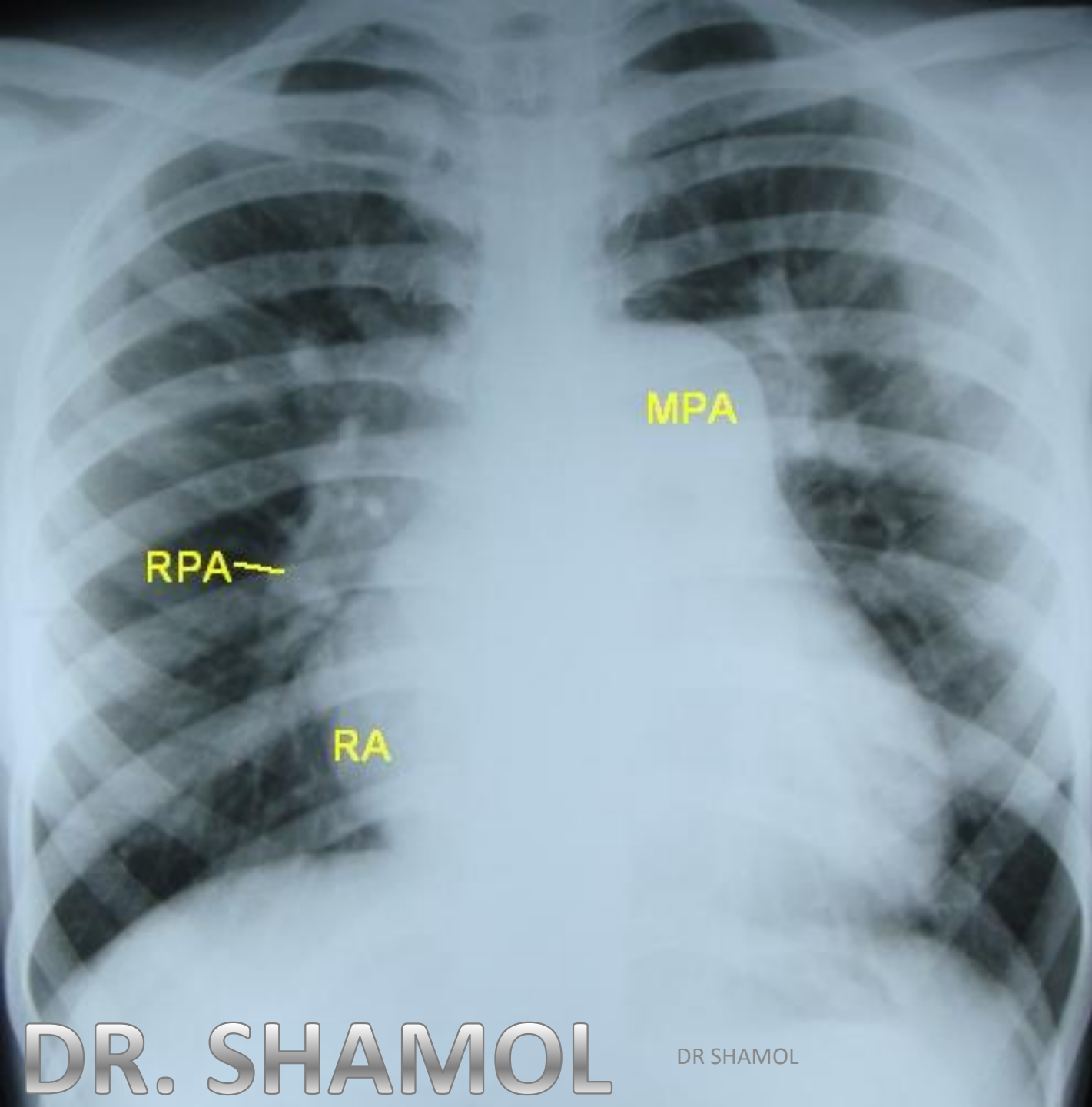
Cardiomegaly  
Rv-type

Plethoric  
lung field

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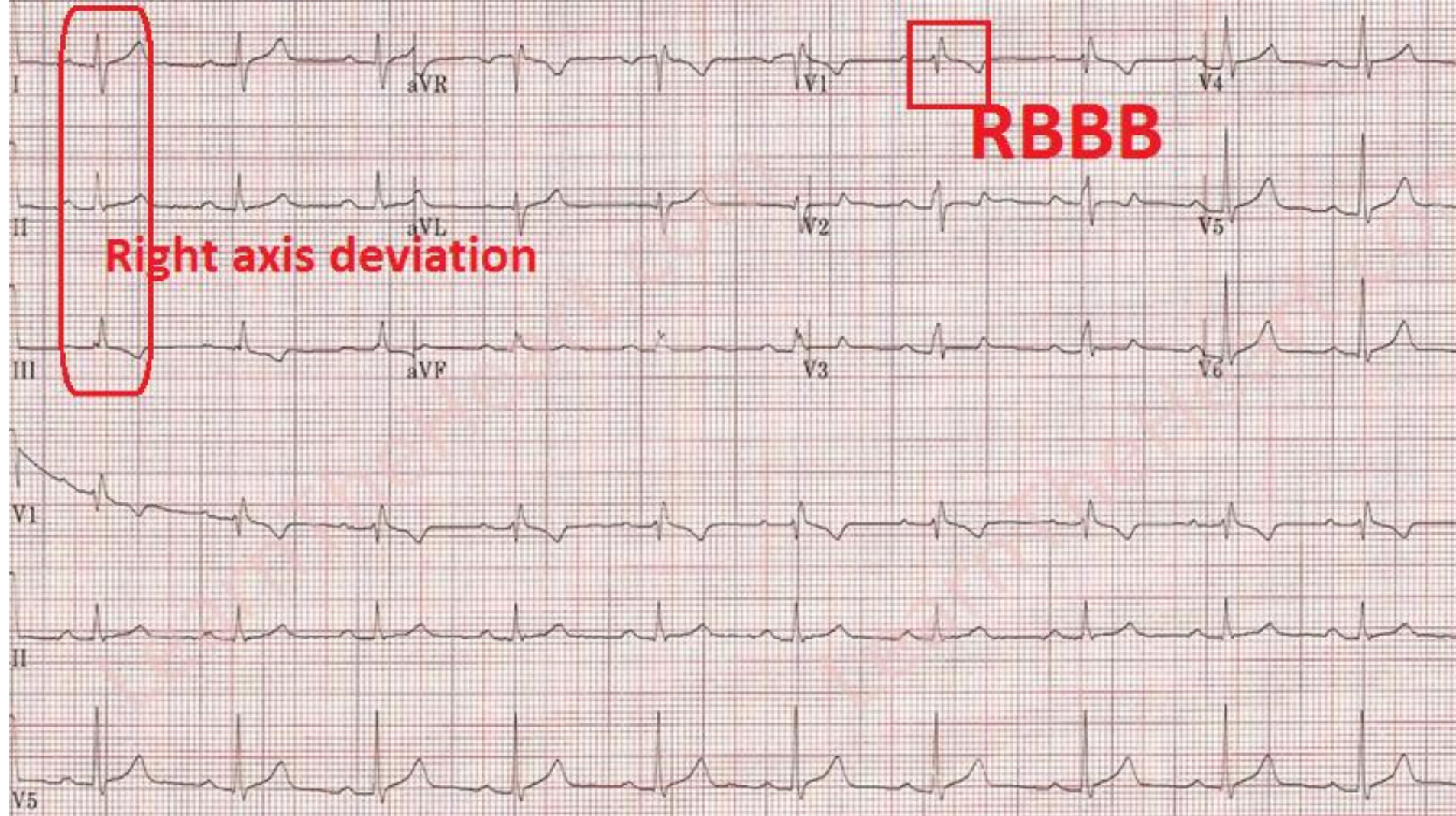
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incomplete right bundle branch

Ostium secundum

RBBB & right axis deviation

ostium primum

RBBB & left axis deviation

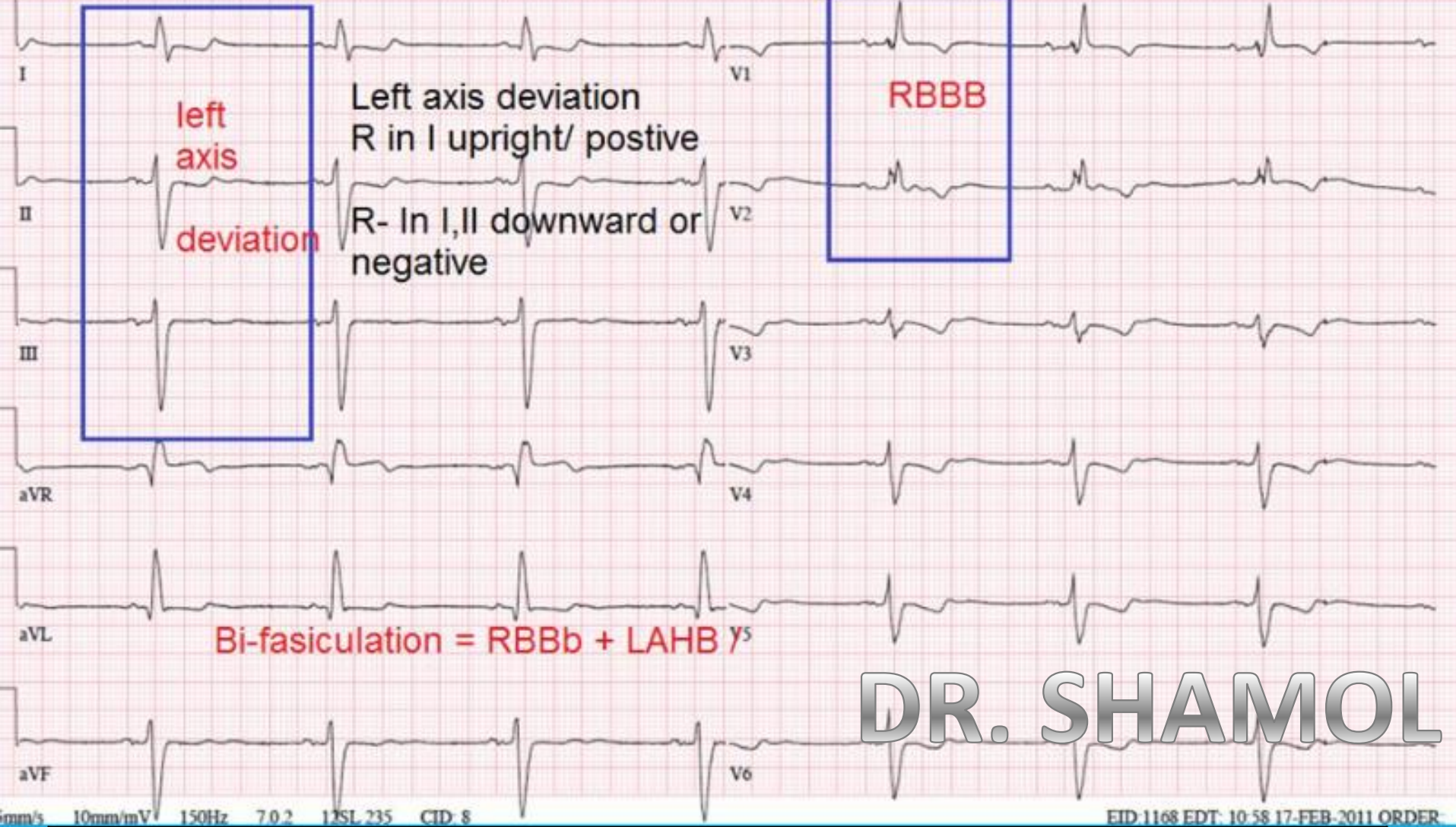
sinus venosus

a junctional or low atrial rhythm  
(inverted P waves in inferior leads)

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incomplete right bundle branch

Ostium secundum

RBBB & right axis deviation

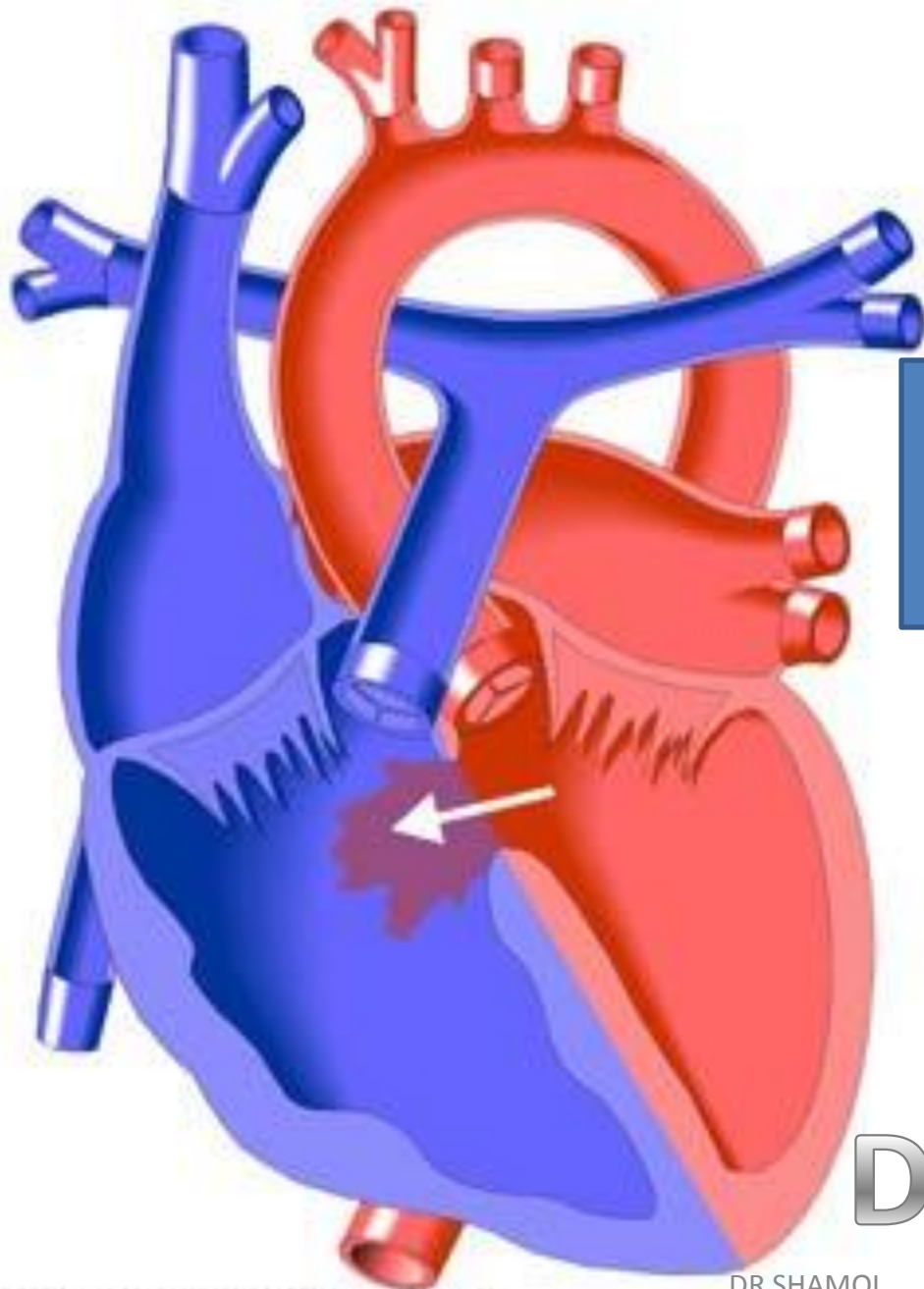
ostium primum

RBBB & left axis deviation

sinus venosus

a junctional or low atrial rhythm  
(inverted P waves in inferior leads)

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V S D

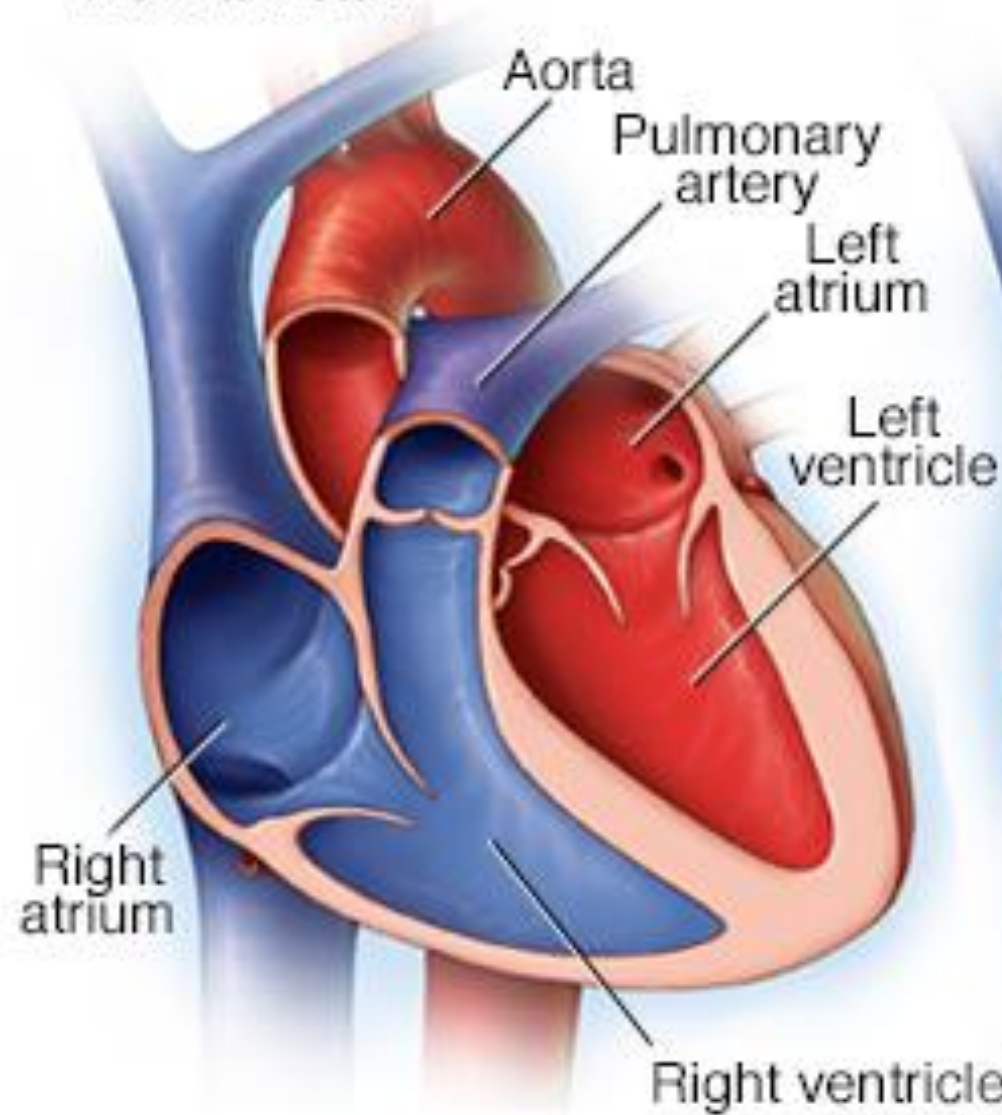
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**Ventricular Septal Defect**

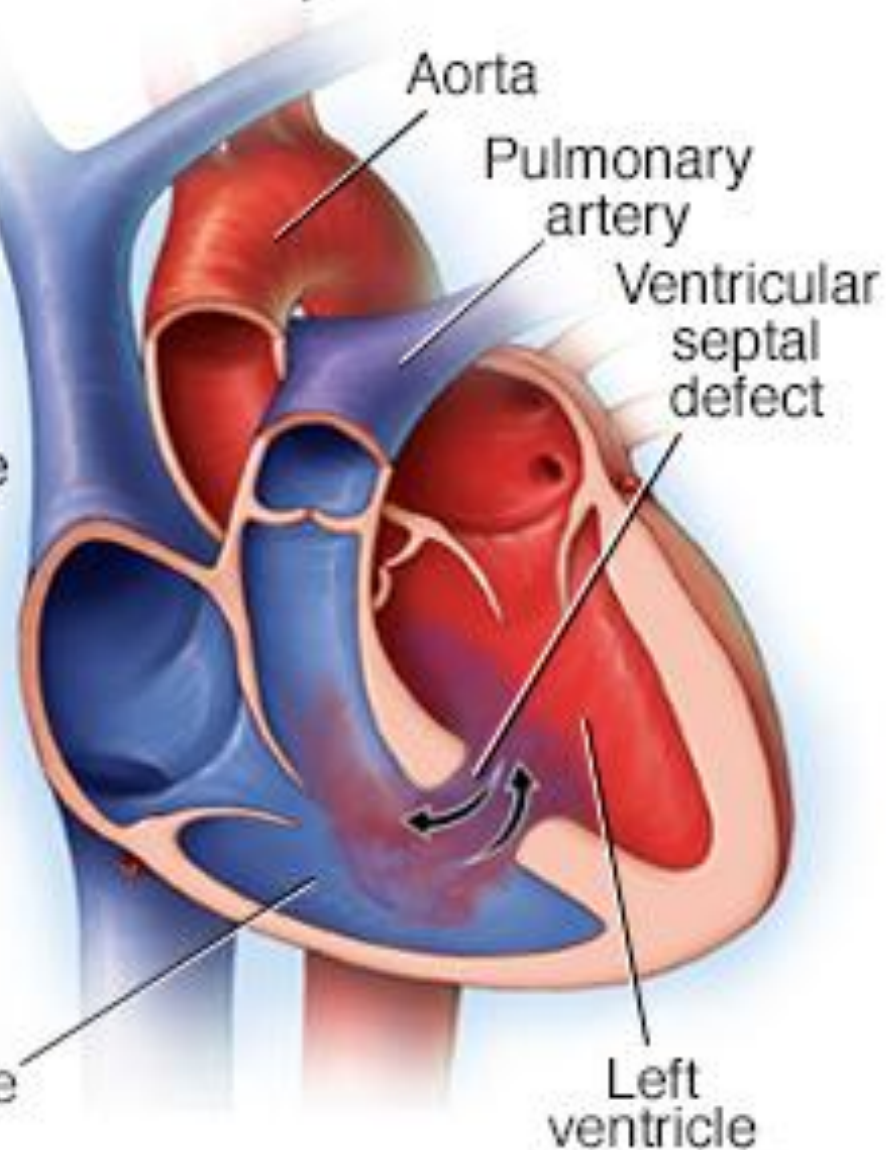
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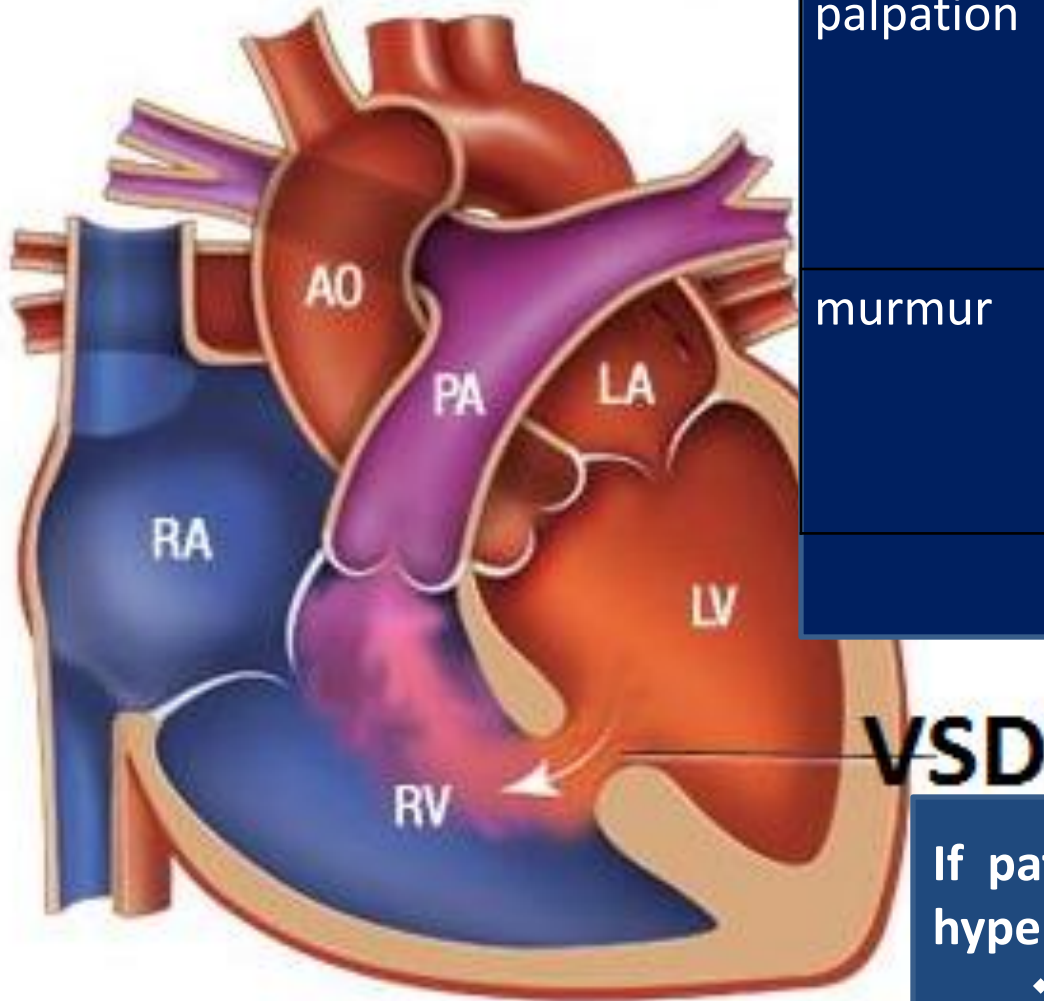


Normal heart



Ventricular septal defect





pulse	normal
palpation	apex beat –thrusting may be shifted if ventricular enlargement
	Thrill at the lower left sternal edge
murmur	pansystolic murmur, usually heard best at the left sternal edge but radiating all over the precordium

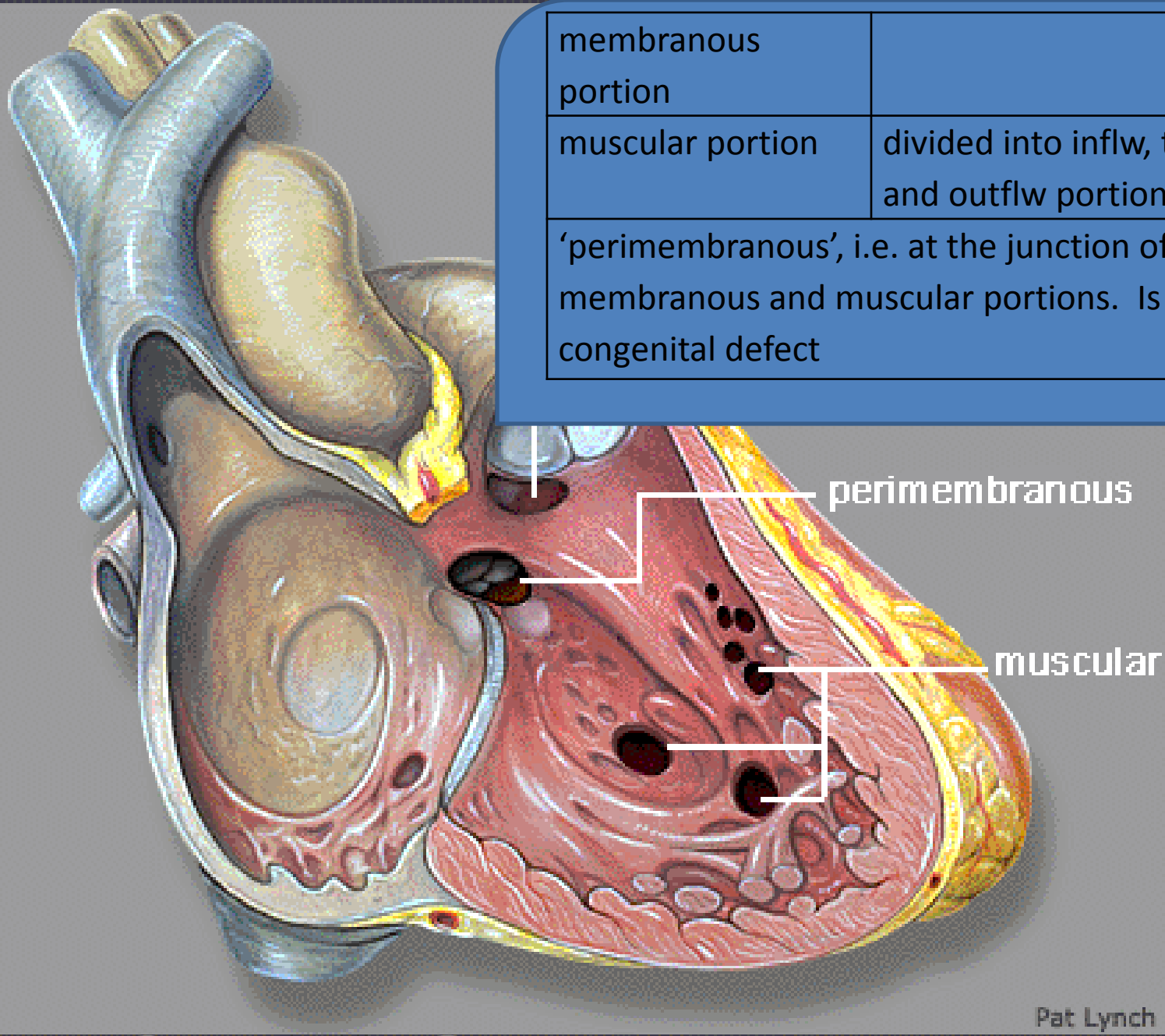
If patient developed pulmonary hypertension then you find following

- ❖ palpable P2
- ❖ Left para-sternal heave

**Auscultation**

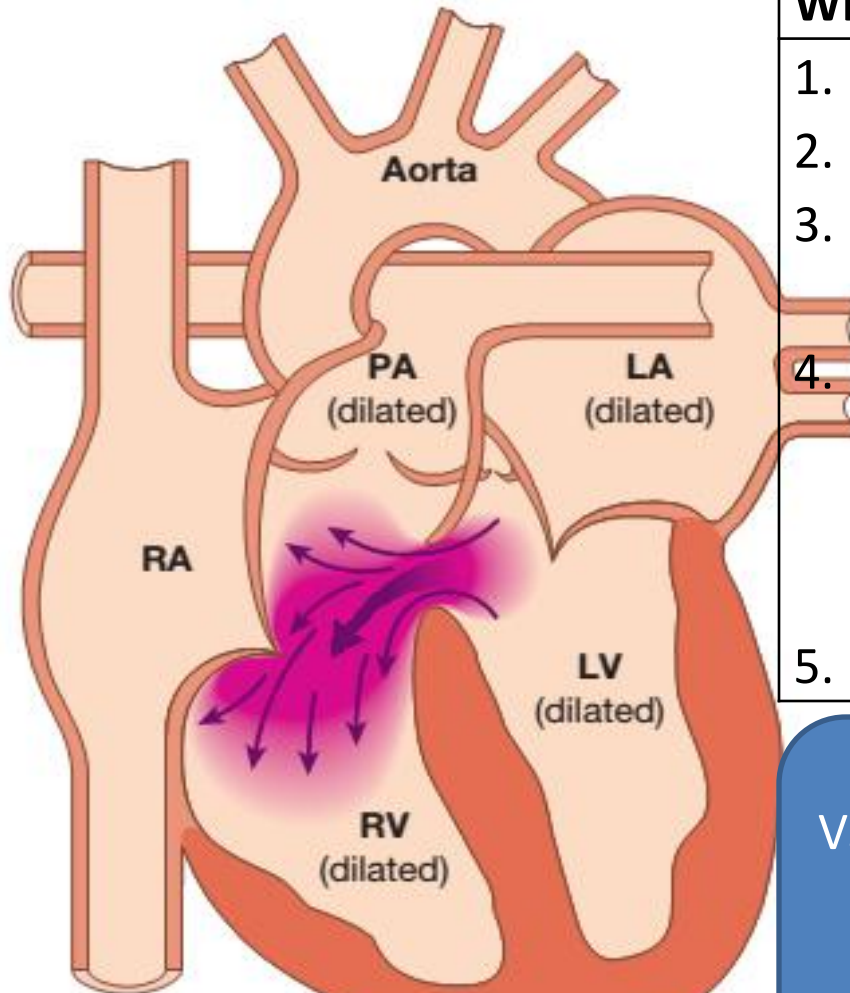
- ❖ loud P2

treatment	If small	Surgery is not needed	
		only follow-up should be done	
		Spontaneous closure may occur in infants if it is in the muscular part	
		Prophylactic penicillin for SBE may be given	
		followed up regularly	
	Moderate to large	Surgical correction is needed if pulmonary to systemic flow ratio >1.5:1	
		Percutaneous transcatheter closure may be done	
When Eisenmenger's syndrome develops		Surgery is contraindicated, as it aggravates right sided heart failure	
		treatments are	Diuretic
			Digoxin in some cases
			Venesection, especially if there is polycythemia
			Heart lung transplantation may





Causes of VSD	congenital heart disease		
	Acquired	result from rupture	complication of acute MI
			from trauma



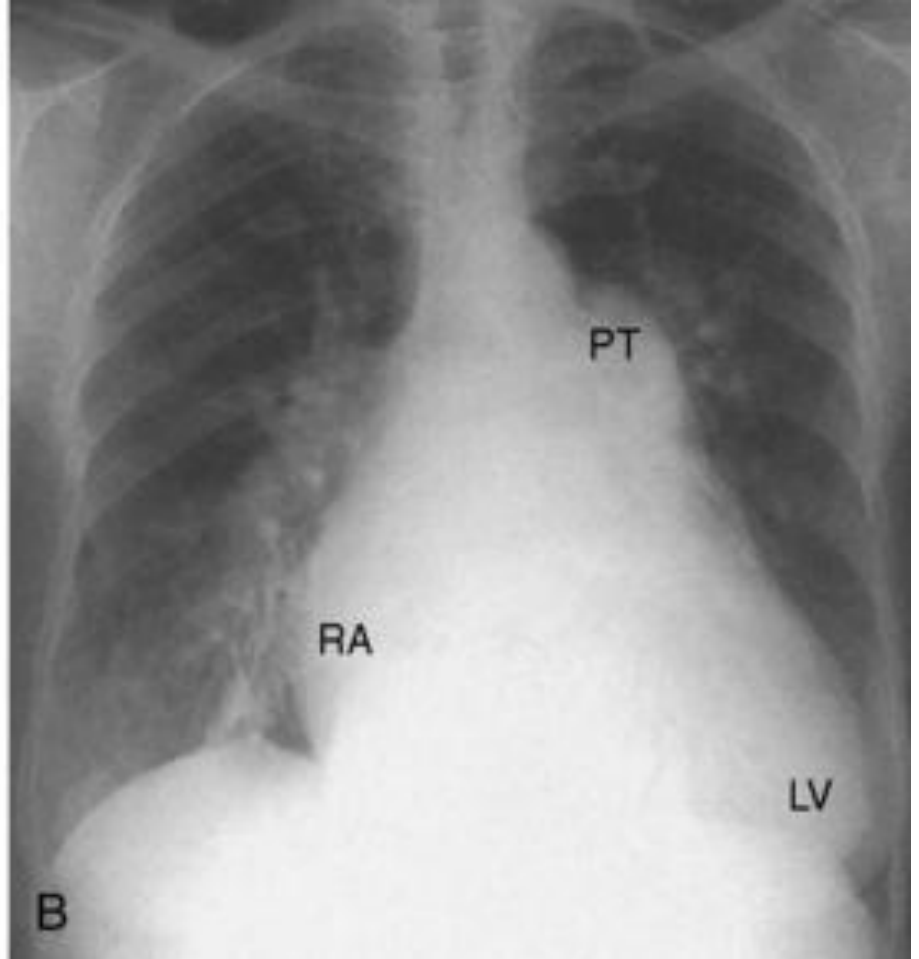
### What is the complication of VSD?

1. Congestive cardiac failure
2. Infective endocarditis
3. Pulmonary hypertension and reversal of shunt (Eisenmenger complex)
4. Right ventricular outflow tract obstruction (muscular infundibular bstruction develops in about of 5% of VSDs)
5. Aortic regurgitation

VSD may be associated with

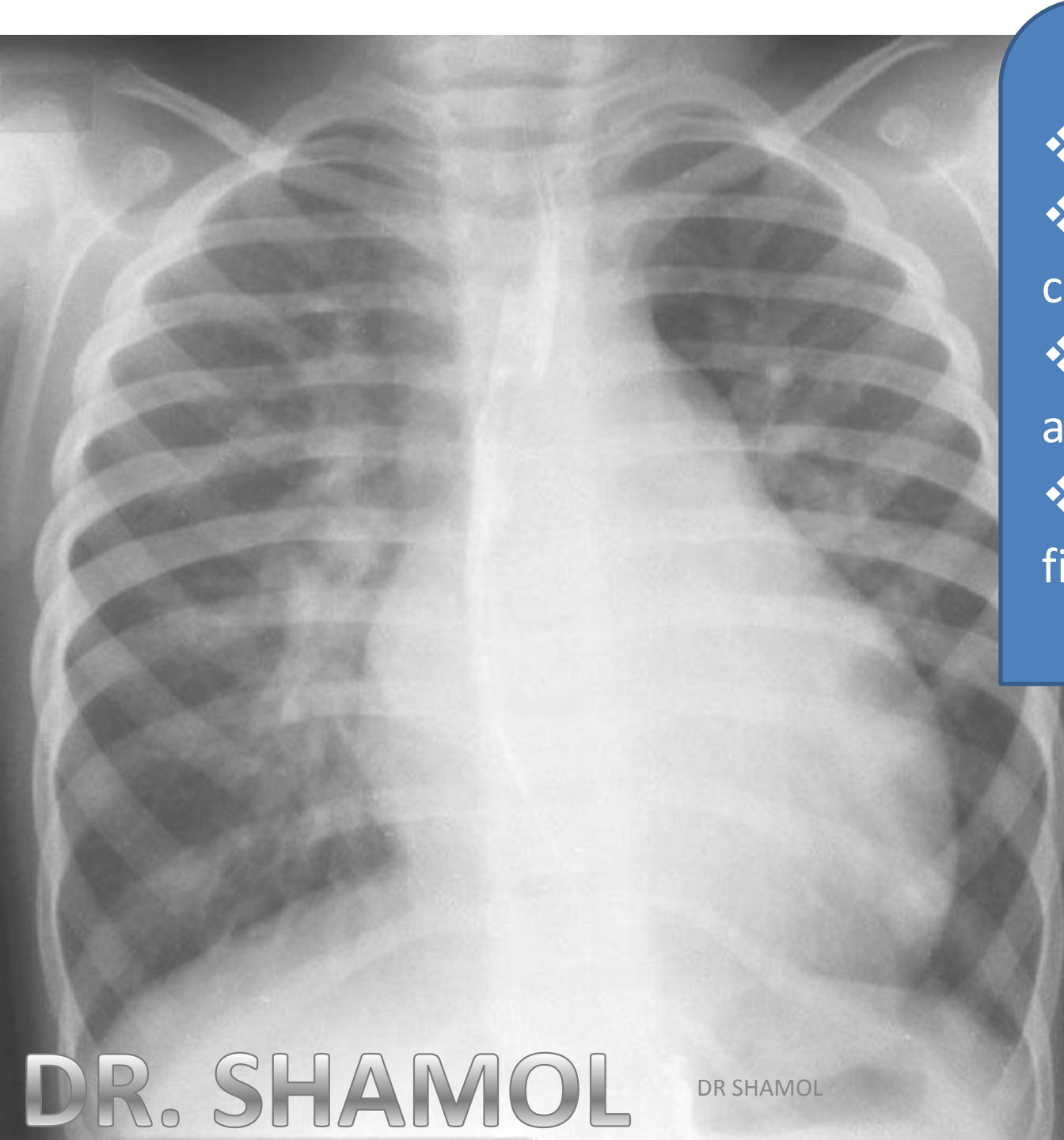
- ❖ Turner's syndrome,
- ❖ Down's syndrome or
- ❖ maternal rubella during pregnancy.

<b>. investigation</b>		
<b>ECG</b>	usually	LVH, biventricular hypertrophy
	if PHT develop	QRS axis shifts to the right and right atrial and ventricular enlargement seen in ECG;
<b>CXR-PA</b>	<ul style="list-style-type: none"> <li>❖ cardiomegaly,</li> <li>❖ large pulmonary conus,</li> <li>❖ large hilar arteries,</li> <li>❖ plethoric lung fields</li> </ul>	
	if pulmonary HTN	<ul style="list-style-type: none"> <li>▪ shows marked enlargement of the proximal pulmonary arteries,</li> <li>▪ rapid tapering of the peripheral pulmonary arteries, and</li> <li>▪ oligemic lung field</li> </ul>
<b>Doppler echocardiography</b>		
<b>Cardiac catheterization and angiography</b>		



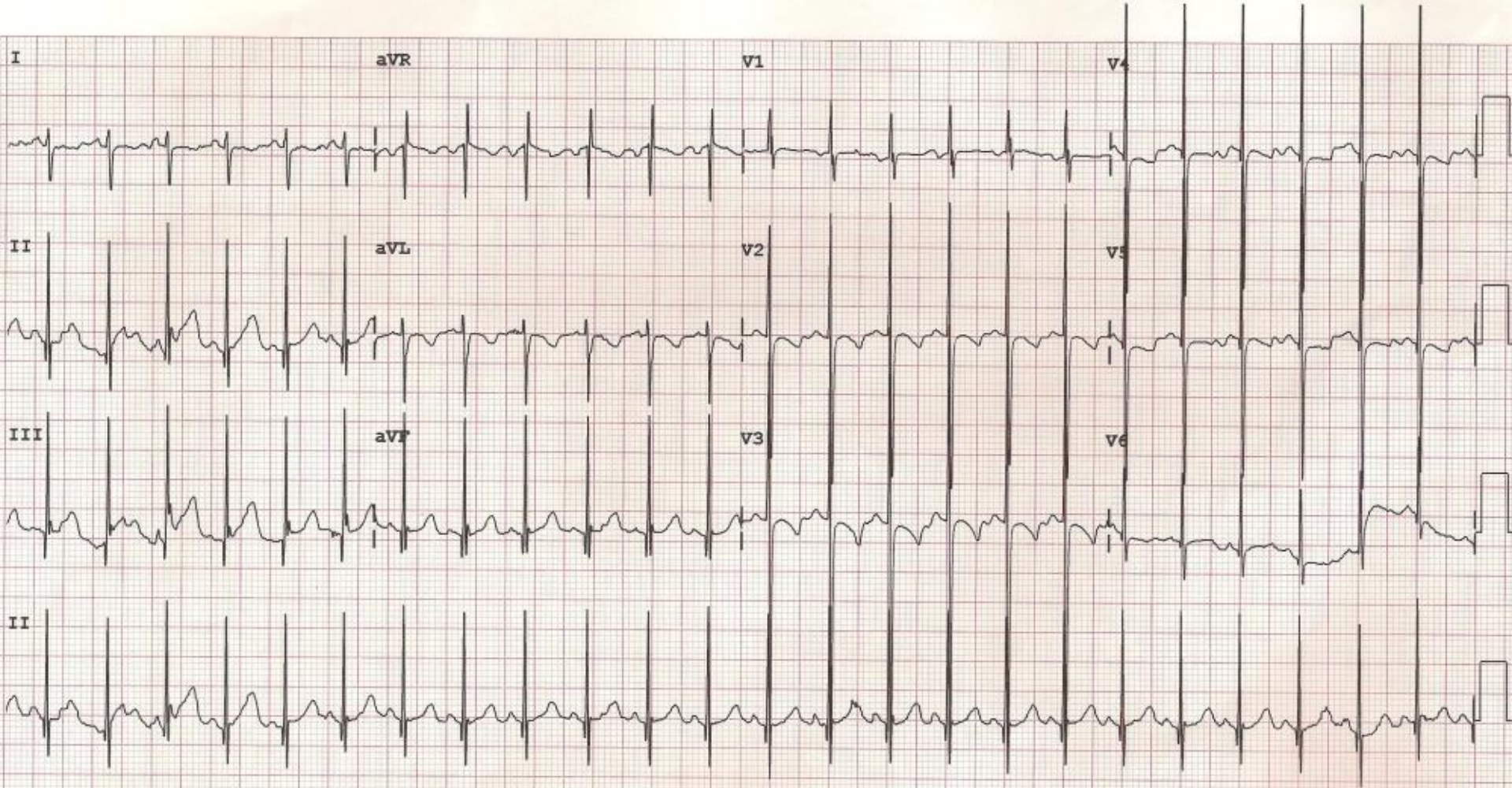
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- ❖ cardiomegaly,
- ❖ large pulmonary conus,
- ❖ large hilar arteries,
- ❖ plethoric lung fields

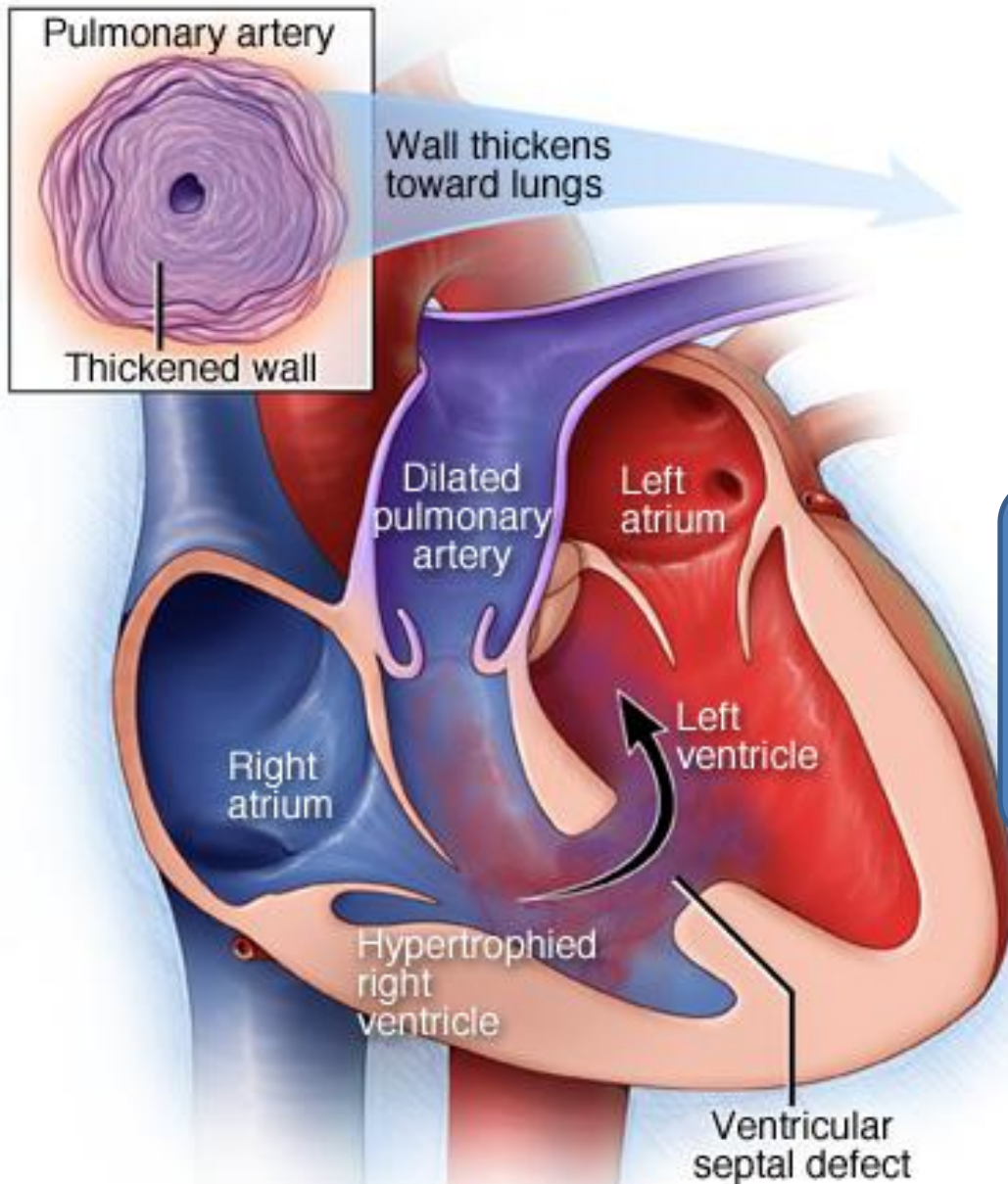




biventricular hypertrophy

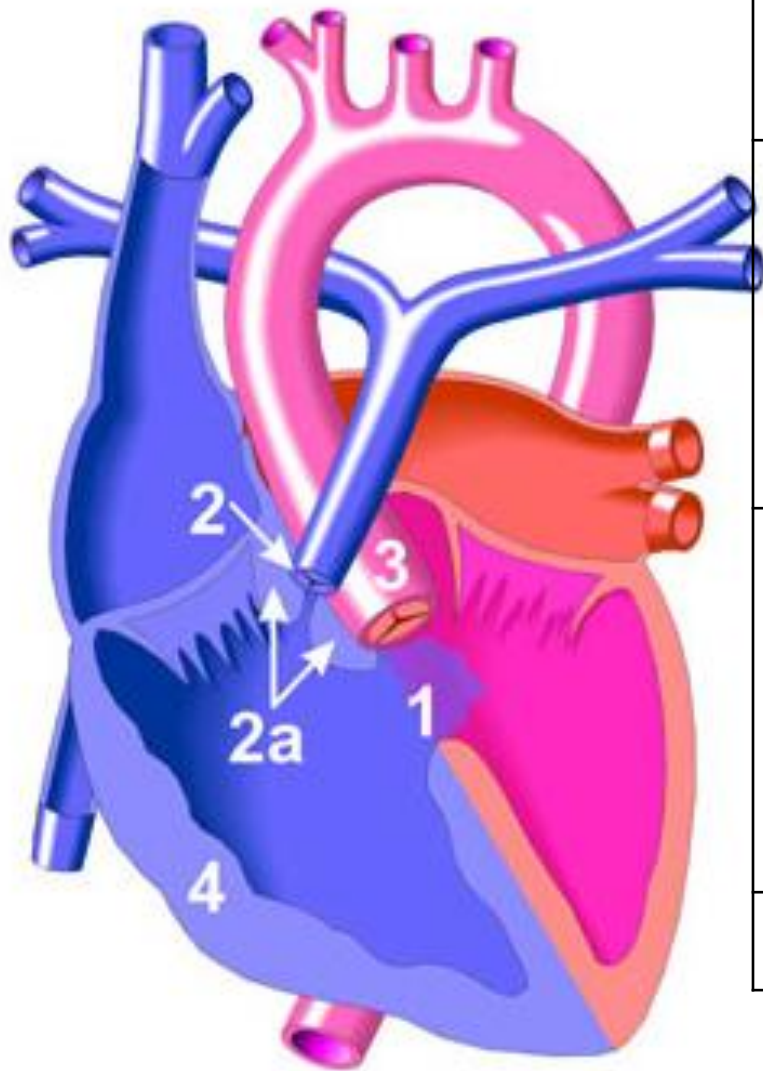
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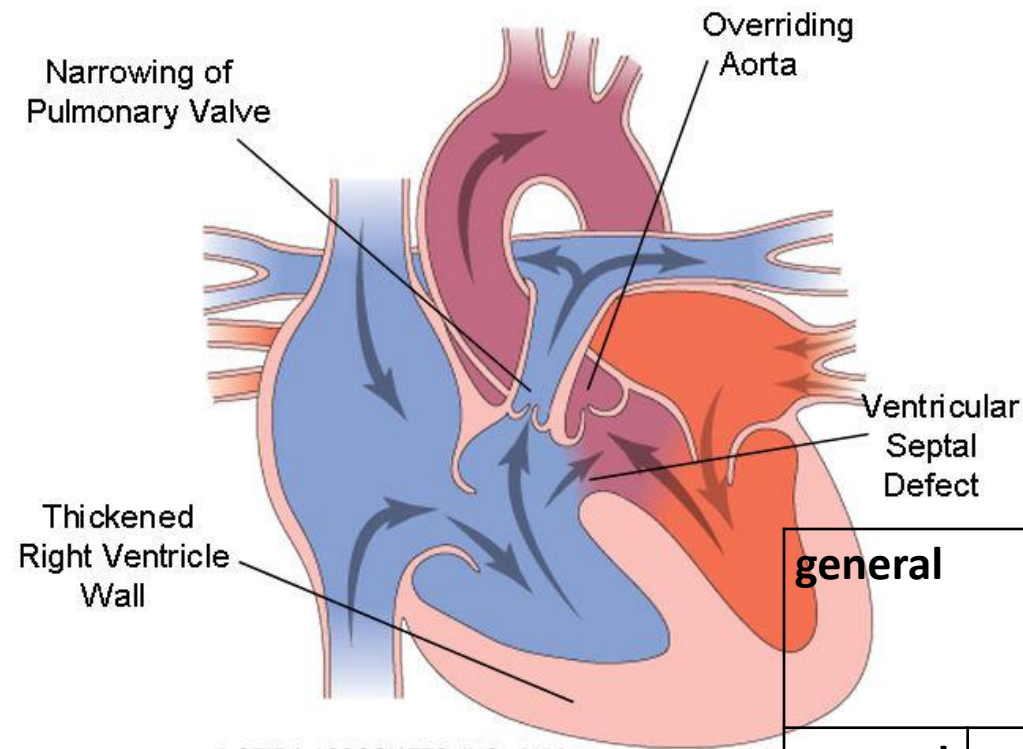
T O F





1	Ventricular septal defect with a right-to-left shunt	
2	pulmonary stenosis (2)with Right ventricular outflow obstruction	subvalvular (infundibular) (2a)
		valvular or
		supravalvular
3	Overriding and dextro-position of aorta(it overriding the ventricular septal defect)	aortic origin— 2/3rd from left ventricle and 1/3rd from right ventricle
4	Right ventricular hypertrophy	

# Tetralogy of Fallot



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## Symptoms

C	Syncope (in 20% of cases)
D	Dyspnea
E	exercise intolerance
F	Fallot's spells relieved by Squatting
G	Growth retardation/ stunting of growth

## general

cyanosis

polycythaemia

digital clubbing

## precordium

palpation

Left parasternal heave

Systolic thrill pulmonary area

auscultation

P2 is soft (or absent)

Ejection systolic murmur heard in the pulmonary area

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**what is the causes of murmur**

**obstructed** Right ventricular flow through the pulmonary stenosed is responsible for systolic murmur

**why murmur was not present at VSD**

pansystolic murmur of VSD may absent because of equalization of pressure between right and left ventricles.

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Children with Tetralogy of Fallot exhibit bluish skin during episodes of crying or feeding.



### What is Fallot's triology?

Atrial septal defect, pulmonary stenosis and right ventricular hypertrophy.

### What is Fallot's pentalogy?

Fallot's tetralogy with associated atrial septal defect.

### What is acyanotic Fallot?

When the TOF is associated with infundibular pulmonary stenosis. Outflow obstruction is mild and there is no cyanosis.

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ADAM

What is 'Fallot's spells or why cyanosis aggravated during exercise ?

We know that The subvalvular / infundibular component of the RV outflow obstruction is dynamic and may increase ( $\uparrow$ infundibular spasm) suddenly under adrenergic Stimulation. Exercise, feeding and crying increase sympathetic stimulation as a result increase right ventricular out flow obstruction. Therefore The affected child suddenly becomes increasingly cyanosed after exertion or crying. this called **Fallot's spells**

Child may be apneic and unconscious. Syncope, seizure, cerebrovascular accident (CVA) or sudden death may occur

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**What is  
Fallot's sign**

How  
squatting  
relieves  
cyanosis?

In squatting position> abdominal aorta and femoral artery are compressed → ↑arterial resistance → ↑increases the pressure in the left ventricle → ↓right to left shunt through VSD → ↑flow through pulmonary artery → so ↓admixture of blood from right and left ventricles →relieves cyanosis → **Fallot's sign**

**DR. SHAMOL**

why cyanosis occur

cyanosis occur when right ventricular pressure rises to equal or exceed left ventricular pressure resulting right-to-left shunt develops. It occur due gradual increase of RV out flow obstruction by pulmonary stenosis (usually valvular / supralvalvular type )

why it absent in neonate

Children are usually cyanosed but this may not be the case in the neonate because at birth pressure in RV is less than the left but due to out flow obstruction

**acyanotic Fallot?**

where pulmonary stenosis is infundibular there Outflow obstruction is mild and there is no cyanosis

## Tetrology of Fallot – Before squatting

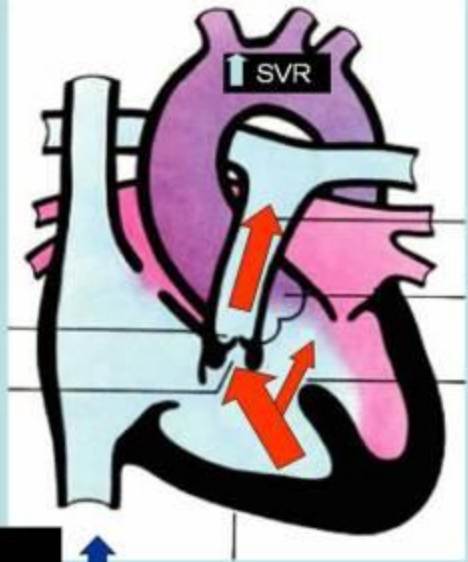


Reduced pulmonary flow

Increased aortic flow

Sustained squatting for 1-2 minutes result in steady increase in venous return, raised systemic vascular resistance.

## Tetrology of Fallot – After squatting

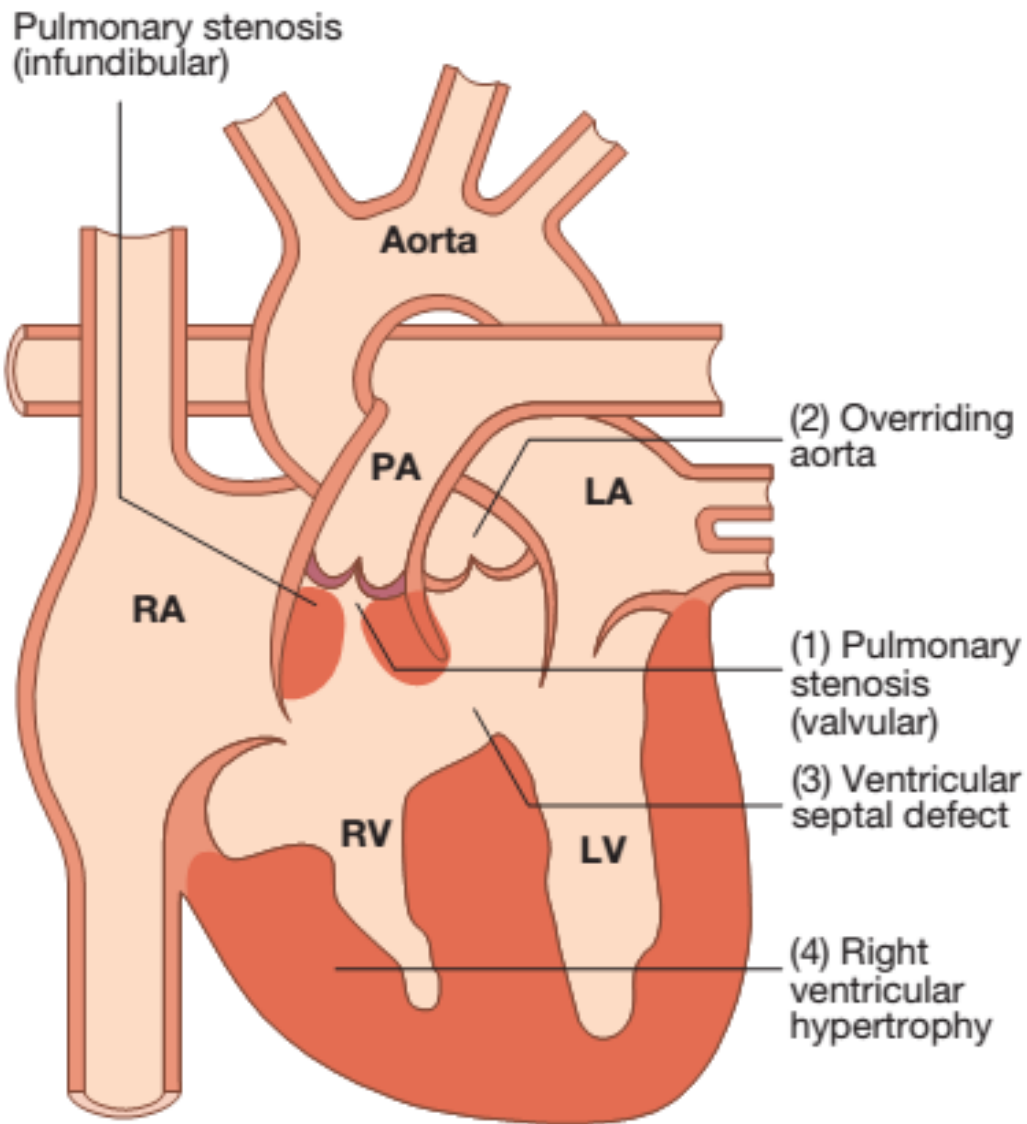


Increased pulmonary flow

Reduced aortic flow

Increased venous return (sustained squatting)

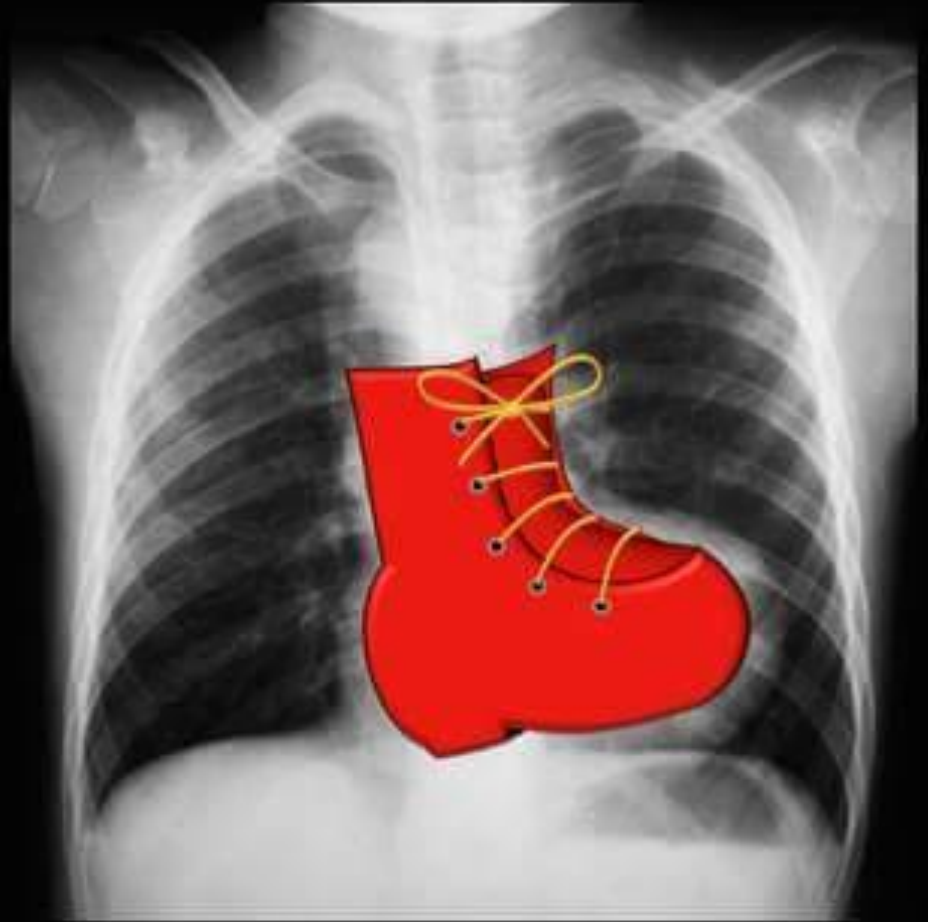
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COMPLICATION		
A	arrhythmia	
C	Cerebral abscess (10% cases)	
C	Coagulopathy	
E	Eisenmenger syndrome	
P	Polycythemia	due to hypoxemia, and may lead to CVD & MI
T	Thrombo-embolic /- Strokes (10%)	Paradoxical emboli.
		thrombotic secondary to erythrocytosis and hyperviscosity

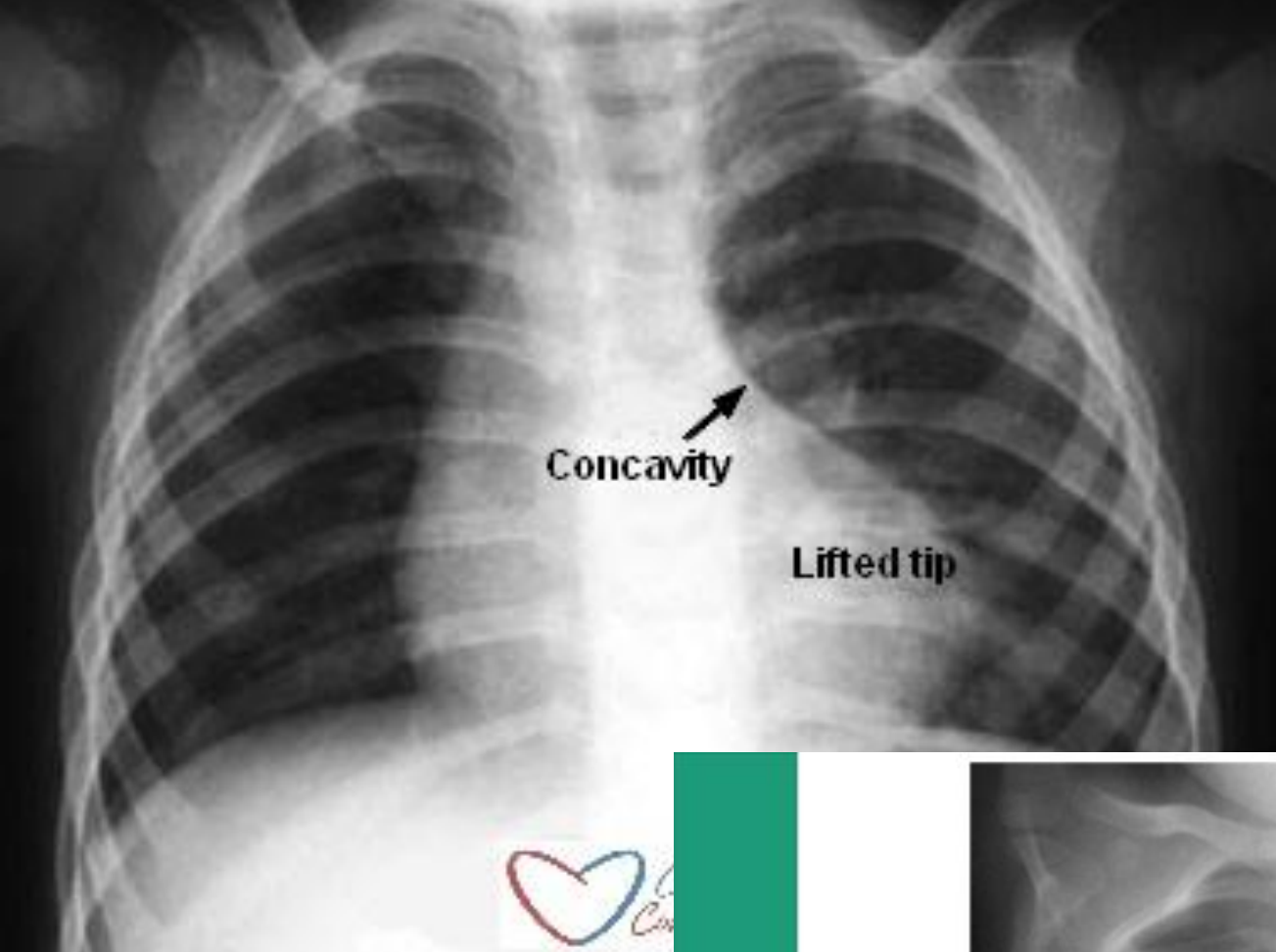


<b>investigation</b>	
<b>ECG shows</b>	The right ventricular hypertrophy
	Right axis deviation
<b>Chest x-ray</b>	‘boot-shaped’ heart.
	pulmonary conus is concave (small pulmonary artery),
	Right-sided aortic arch (in 30% of cases).
	Enlarged RV(prominent elevated apex),
	oligemic lung (Decreased pulmonary vasculature )
<b>Echocardiography</b>	is diagnostic
	demonstrates that the aorta is not continuous with the anterior ventricular septum

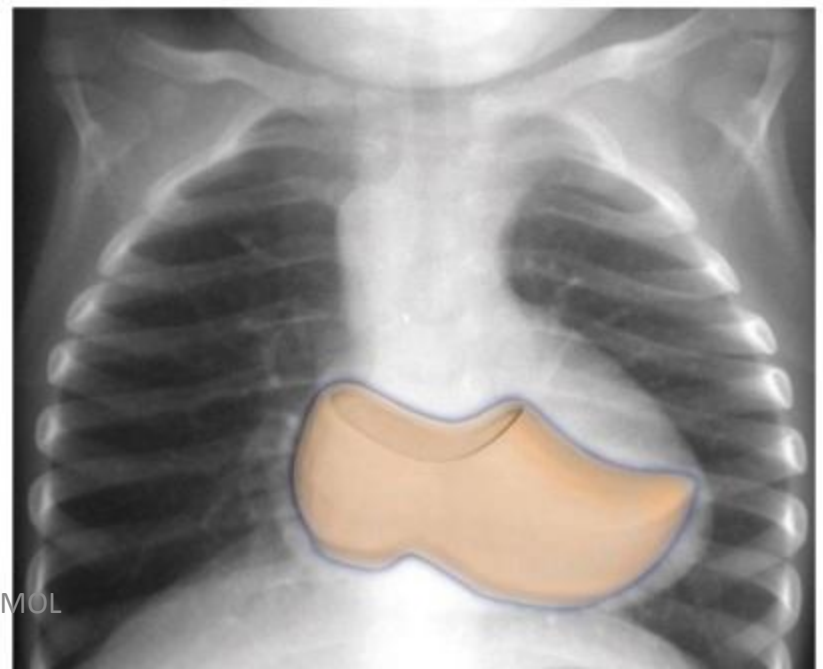


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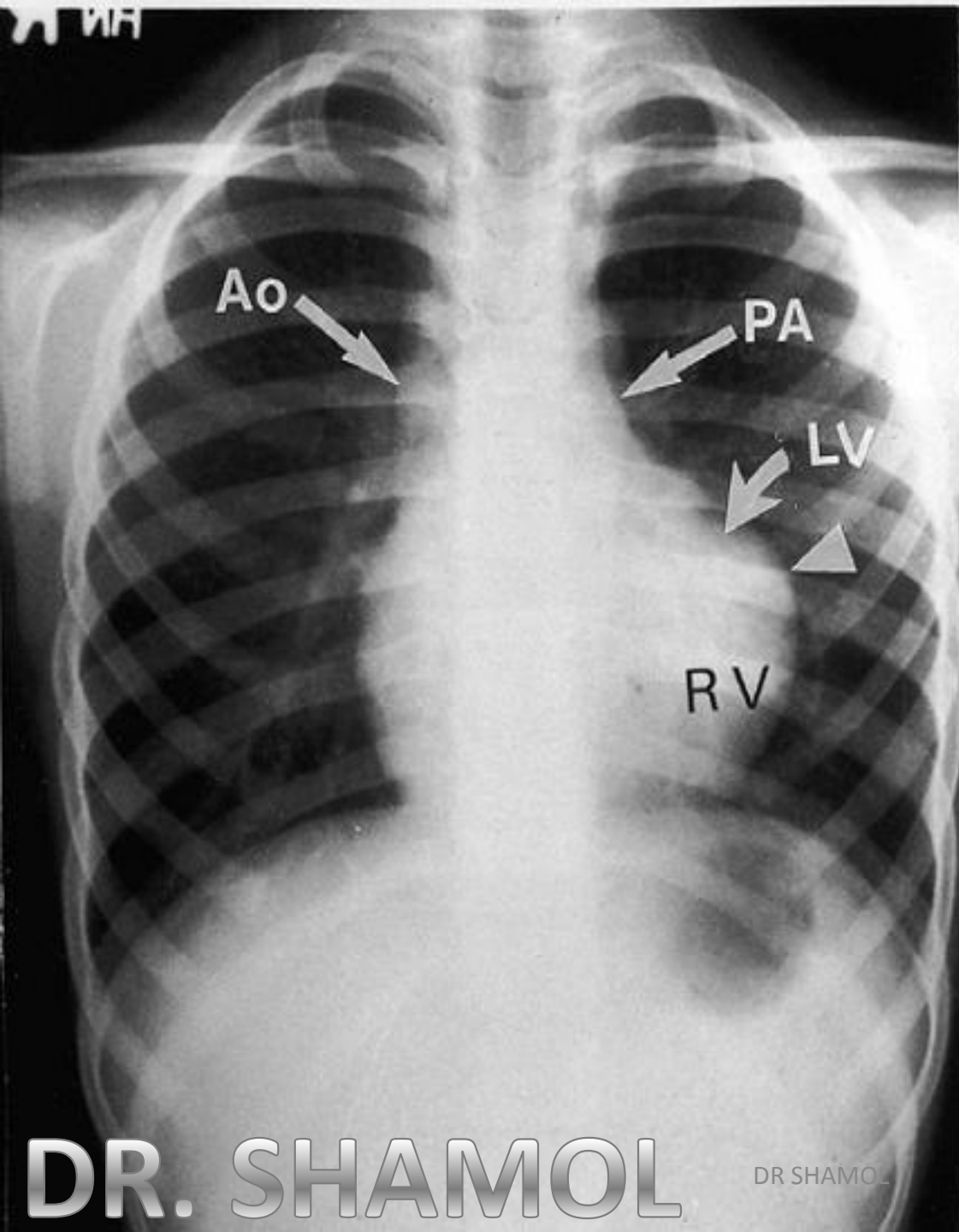


TOF



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**left ventricle (LV)**  
**is small and underfilled and lies**  
**superior to a relatively horizontal**  
**ventricular septum**

an elevated interventricular sulcus  
(arrowhead) inferior to which lies the  
concentrically hypertrophied apex  
forming right ventricle (RV).

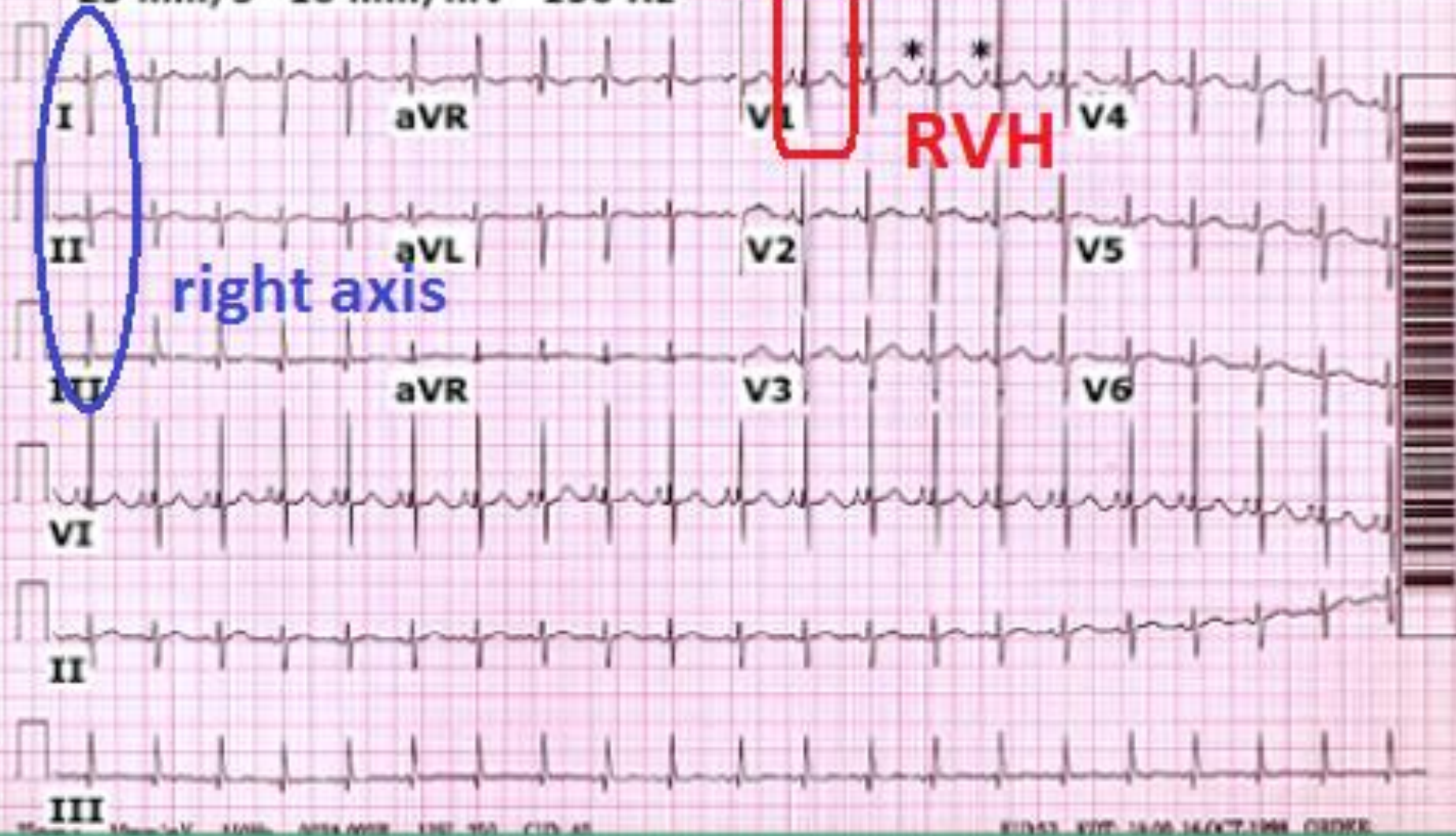
The ascending aorta (Ao) is  
prominent

the main pulmonary artery segment  
(PA) is concave,

the lungs are oligoemic



25 mm/s 10 mm/mV 150 Hz



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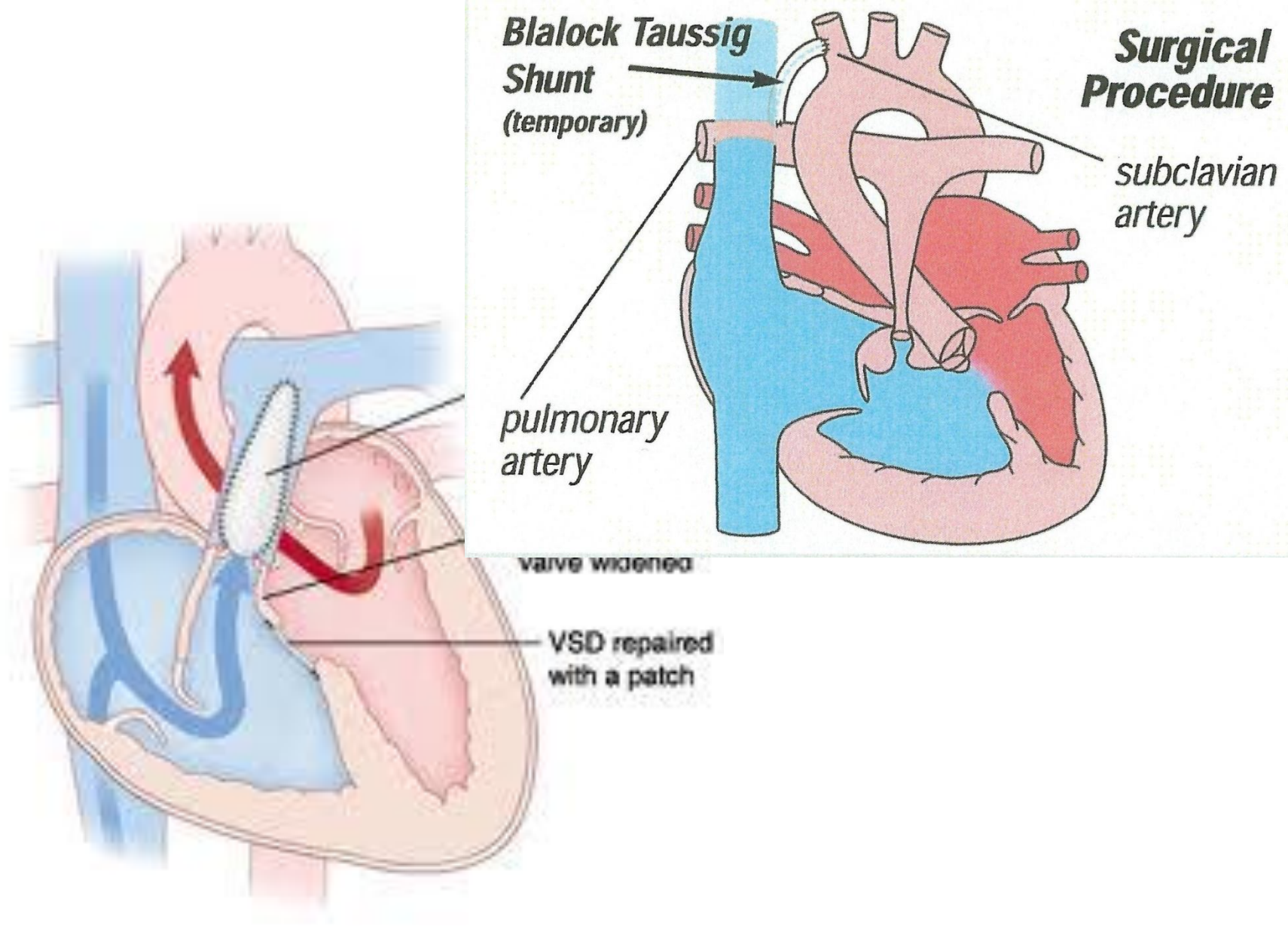
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<b>Definitive treatment</b> .	Surgical correction	
	Primary surgery	done prior to 5 years of age
		Surgical relieve of pulmonary stenosis and closure of VSD
	temporarily palliative surgery	done If the pulmonary arteries are too hypoplastic
		Corrective surgery is done later on.
<b>to prevent infective endocarditis →</b> Prophylactic antibiotic		

### What is Blalock-Taussig shunt?

. Blalock-Taussig shunt is the anastomosis between left subclavian artery with left pulmonary artery. This improves pulmonary blood flw and pulmonary artery development, and may facilitate defiitive surgery later on

How to treat during cyanotic spell	Knee-chest position of child
	High concentration of o2
	Injection morphine or diamorphine (it relaxes right ventricular outflow obstruction)
	β-blocker may be used
	If medical therapy fails, emergency surgical shunt may be considered

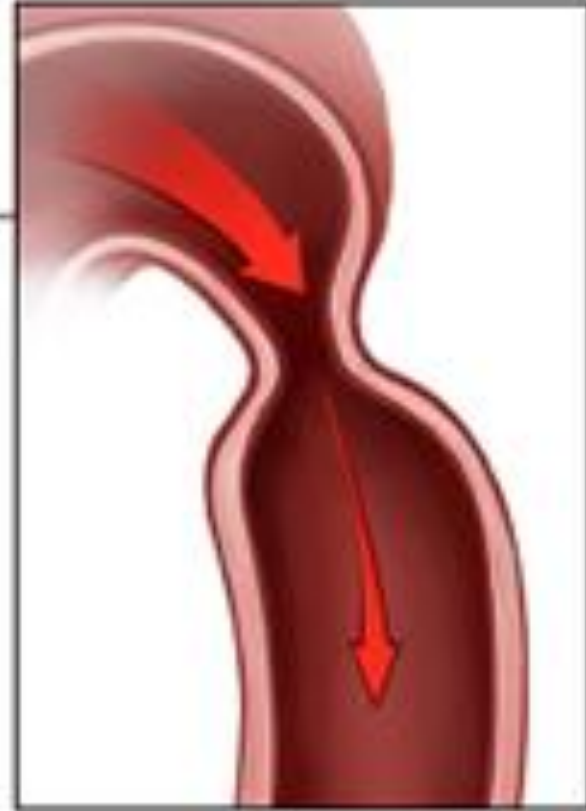
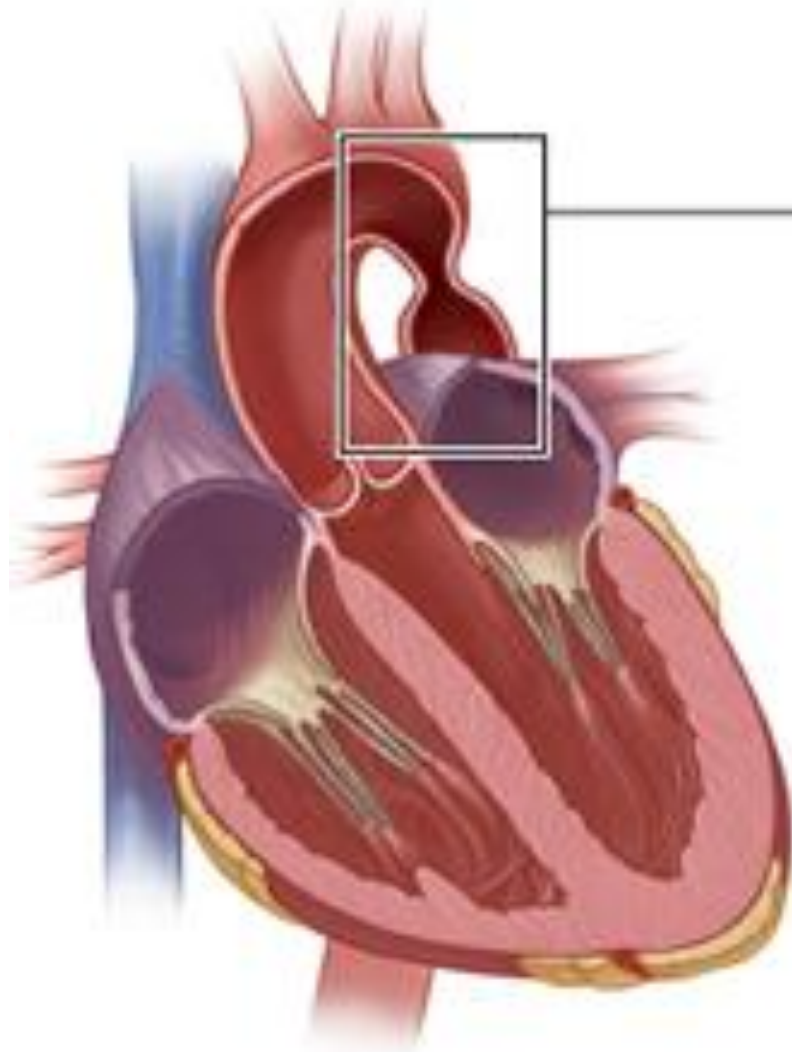


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# Coarctation of the aorta



Narrowed aorta reduces  
blood flow to body



<b>what is Coarctation of the aorta ?</b>	
Narrowing of the aorta just below the origin of the left subclavian artery (occurs in the region where the ductus arteriosus joins the aorta)	
epidemiology	males : female :2:1
usually	congenital
Acquired coarctation	following trauma
	complication of a progressive arteritis (Takayasu's disease)
association (Davidson )	bicuspid aortic valve
	berry' aneurysms of the cerebral circulation
Genetic disease have coarctation	turner syndrome

# upper limb BP > Lower limb



symptoms	Asymptomatic usually
	Symptoms of hypertension:
	<ul style="list-style-type: none"> <li>• headache,</li> <li>• epistaxis,</li> <li>• dizziness, and</li> <li>• palpitations</li> </ul>
	Claudication and cold leg -- decreased circulation in the lower part of the body
	symptoms of heart failure or aortic dissection.
	Women with coarctation are at particularly high risk for aortic dissection during pregnancy

<b>Pulse</b>	high volume in upper limb
	radio-femoral delay – (if coarctation distal to left subclavian artery )
	radio-radial delay – (if coarctation proximal to left subclavian artery )
	Carotid pulse: High volume and vigorous
	There is visible suprasternal, right carotid pulse and supraclavicular pulsation
<b>BP</b>	systolic arterial pressure is higher in the arms than in the legs
	but the diastolic pressures are similar
	a widened pulse pressure is present in the arms

High blood pressure before point of coarctation

Upper limb

Lower limb

Low blood pressure beyond point of coarctation

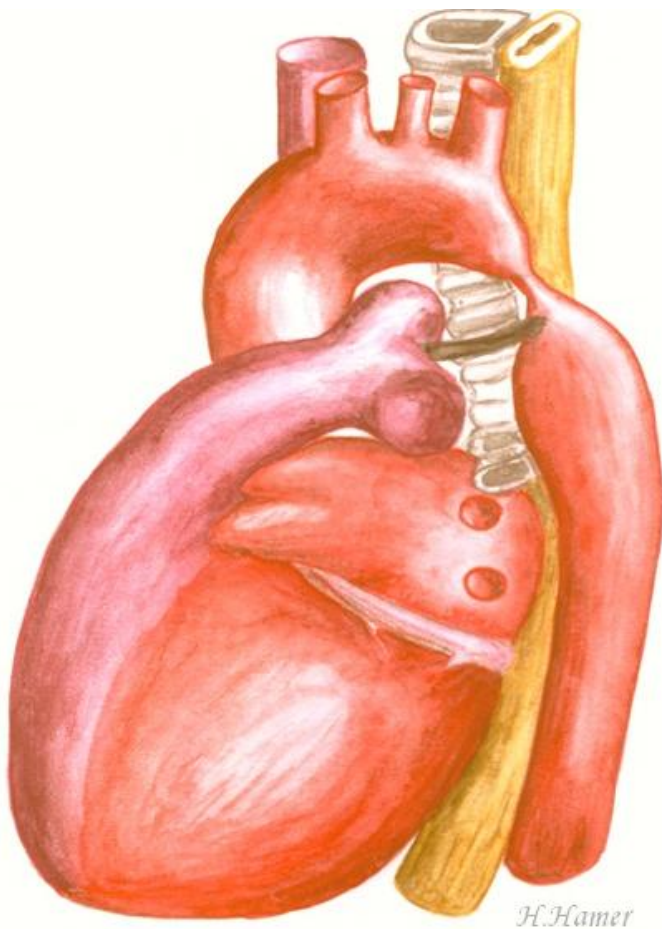
Radio-femoral delay

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Coarctation of the aorta



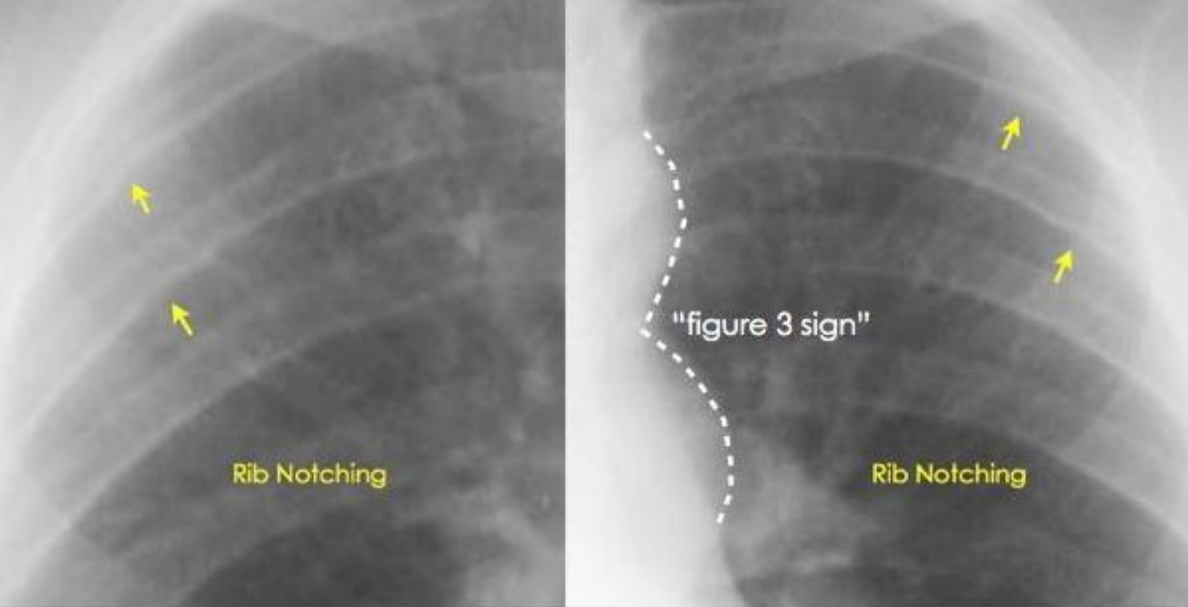
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ADAM



inspection	Visible cardiac impulse	
	Visible dilated tortuous artery around the scapula, anterior axilla and over the left sternal border	
	collateral vessels are best seen by sitting and bending forward, with arm hanging by the side	
palpation	apex beat is shifted & heaving in nature	
	thrill over the collateral vessels and suprasternal notch	
Auscultation	A systolic murmur intercostal space close to the sternum and better heard in 4th intercostal space posteriorly (site of coarctation).	
	an ejection click and systolic murmur in the aortic area due to a bicuspid aortic valve	
	EDM (bicuspid aortic valve or dilatation of aortic valve due to aneurysm, causing AR).	
	localised bruits	As a result of the aortic narrowing, collaterals form over the periscapular, internal mammary and intercostal arteries, and may result in bruit



<b>The ECG</b>	left ventricular hypertrophy	
<b>chest X-ray</b>	early childhood is often normal	
	changes in the contour of the aorta <b>3' sign</b>	indentation of the descending aorta causes characteristic '3' sign on a chest radiograph
		The upper bulge is formed by dilatation of the left subclavian artery
		the sharp indentation is the site of the coarctation
		the lower bulge is called the poststenotic dilatation of the aorta.
	aortic knuckle	Poorly developed aortic knuckle (or elongated aortic knuckle), cardiomegaly, post-stenotic dilatation of aorta
	symmetric bilateral rib notching	notching of the middle part posteriorly ribs from enlargement of intercostal arteries from 3rd rib downwards (1st and 2nd ribs are not affected, because intercostal arteries here arise from subclavian artery above the constriction)—occur due to increased collateral circulation
<b>Echocardiography</b>	may visualize the coarctation	
<b>Doppler examination</b>	estimate of the transcoarctation pressure gradient	
<b>MRI and contrast aortography</b>		investigation of choice



### What causes rib notching?

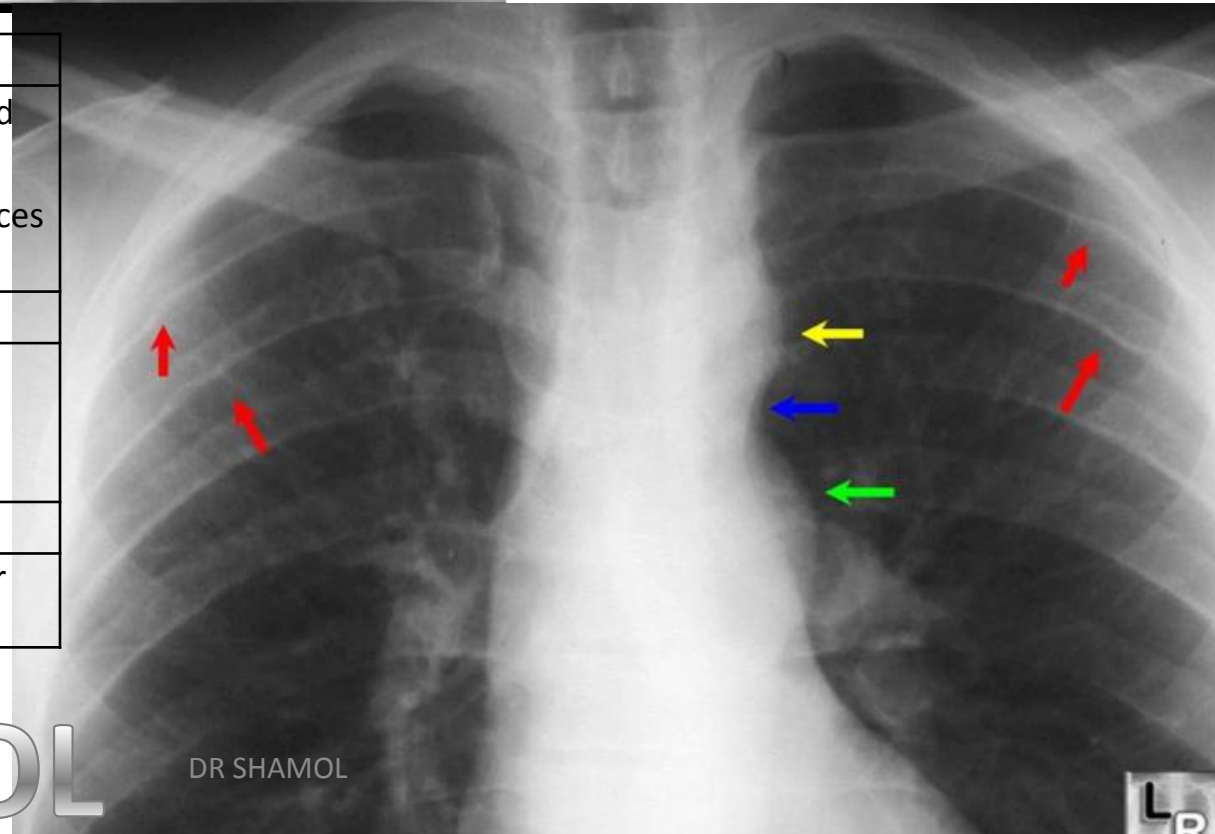
Collateral flow through dilated, tortuous and pulsatile posterior intercostals arteries typically causes notching on the undersurfaces of the posterior portions of the ribs

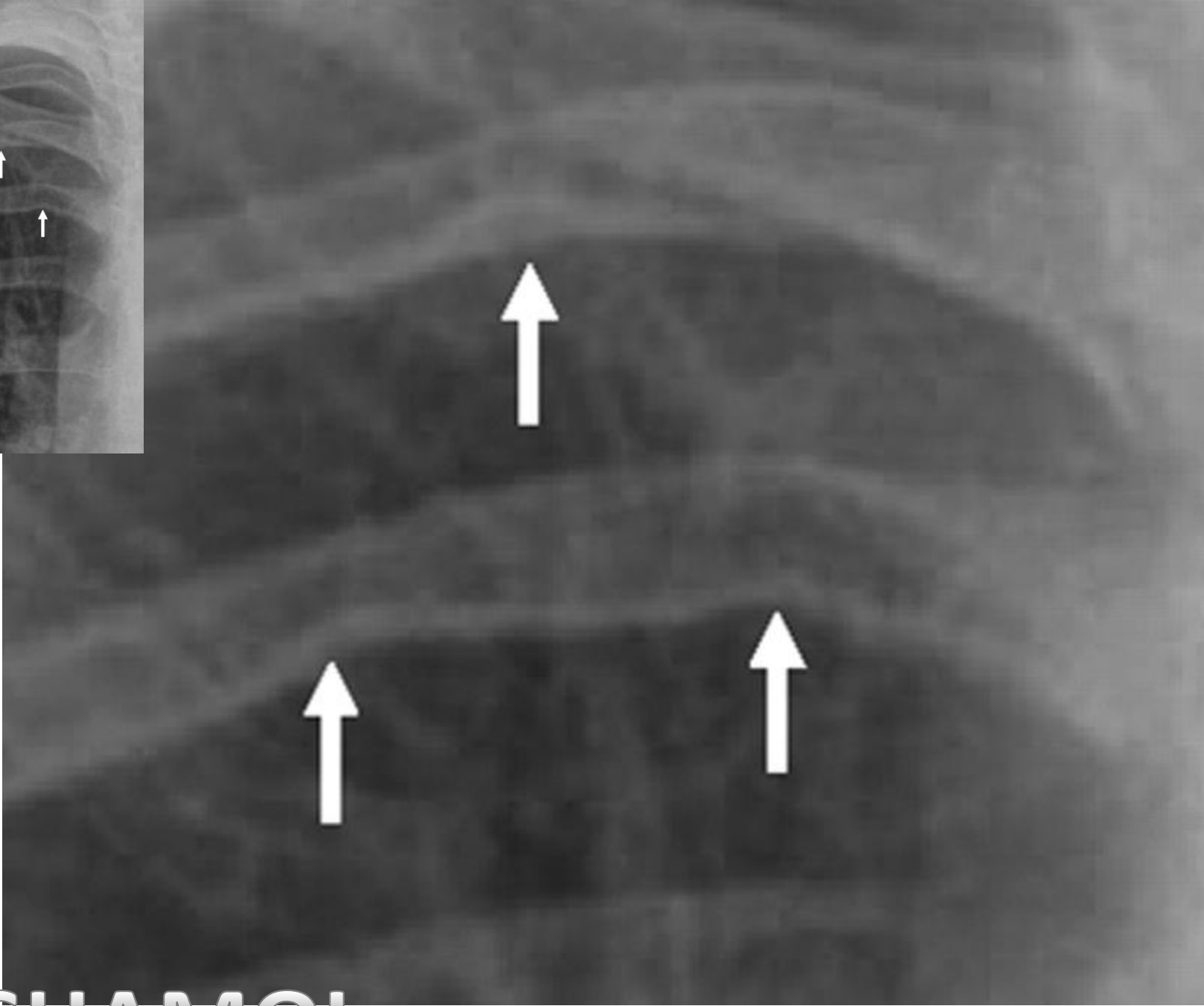
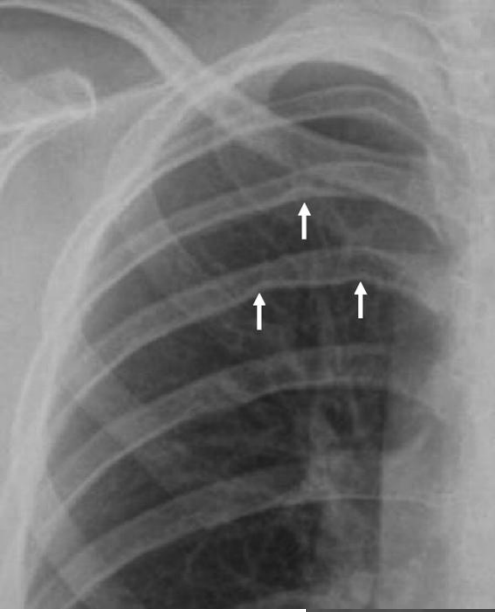
### why not anterior part of rib

The anterior parts of the ribs are spared because the anterior Intercostal arteries do not run in the costal grooves

### in which rib

Notching is seldom found above the third or below the ninth rib and rarely





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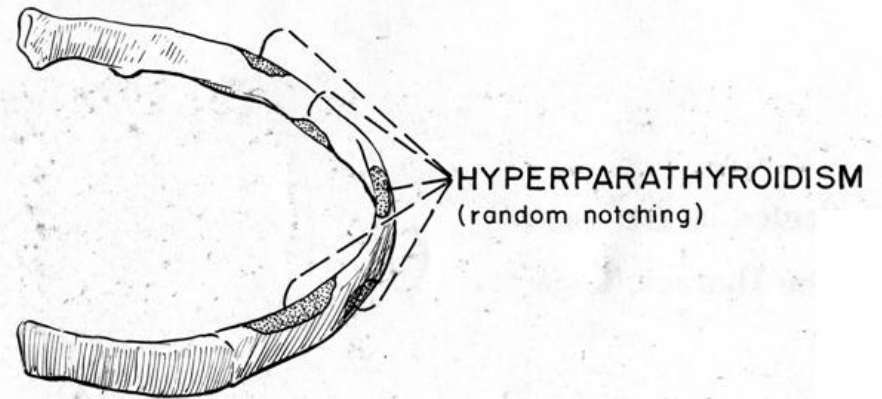
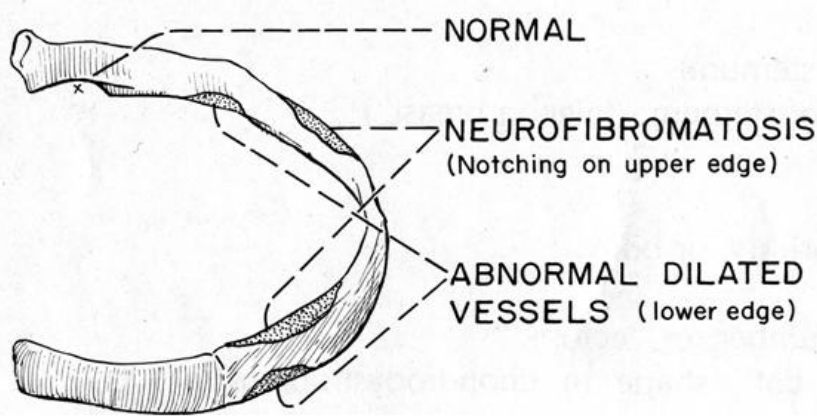
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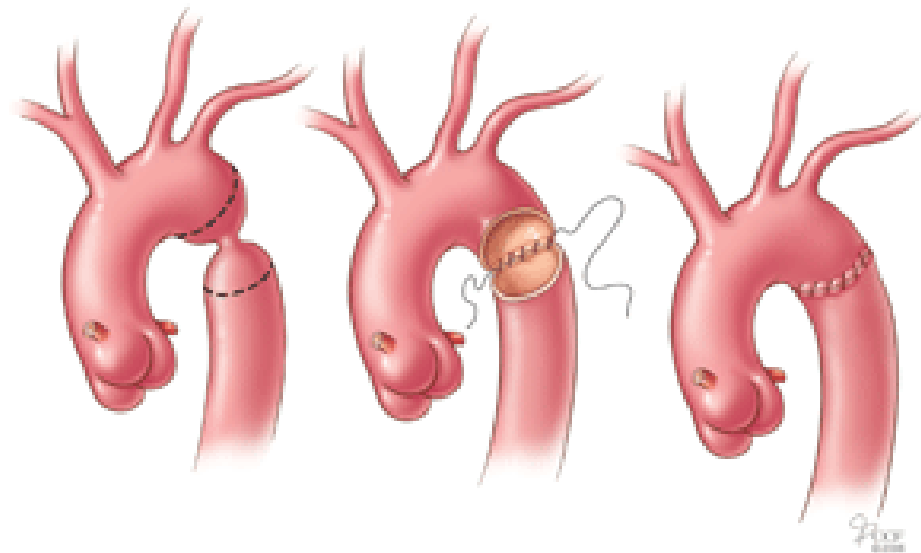
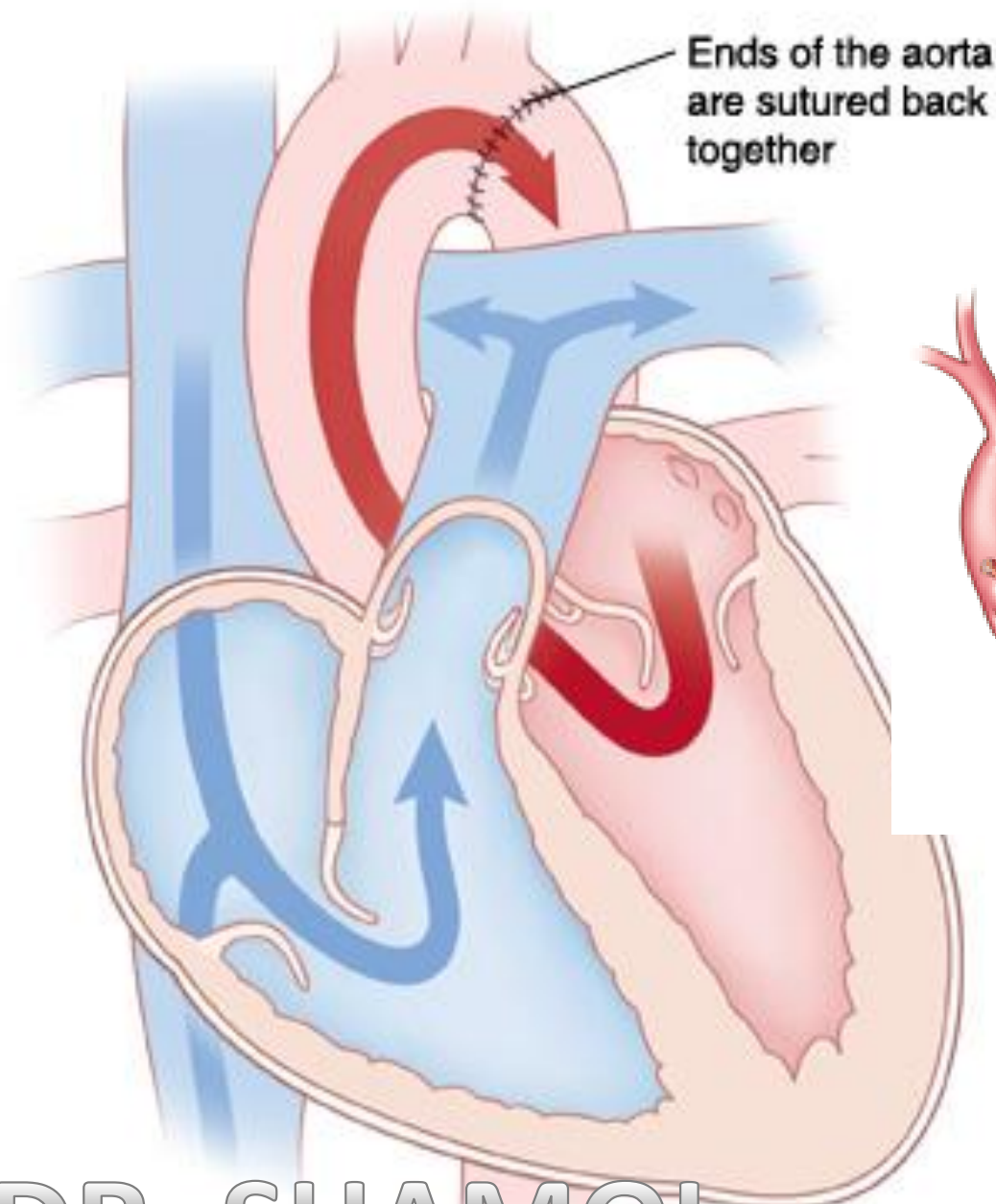
*Rib Notching*

### **unilateral rib notching**

- ❖ Coarctation of aorta (before the origin of left subclavian artery)
- ❖ Blalock-Taussig shunt (iatrogenic, done in Fallot's tetralogy)
- ❖ Subclavian artery obstruction
- ❖ Neurofiromatosis
- ❖ Congenital.

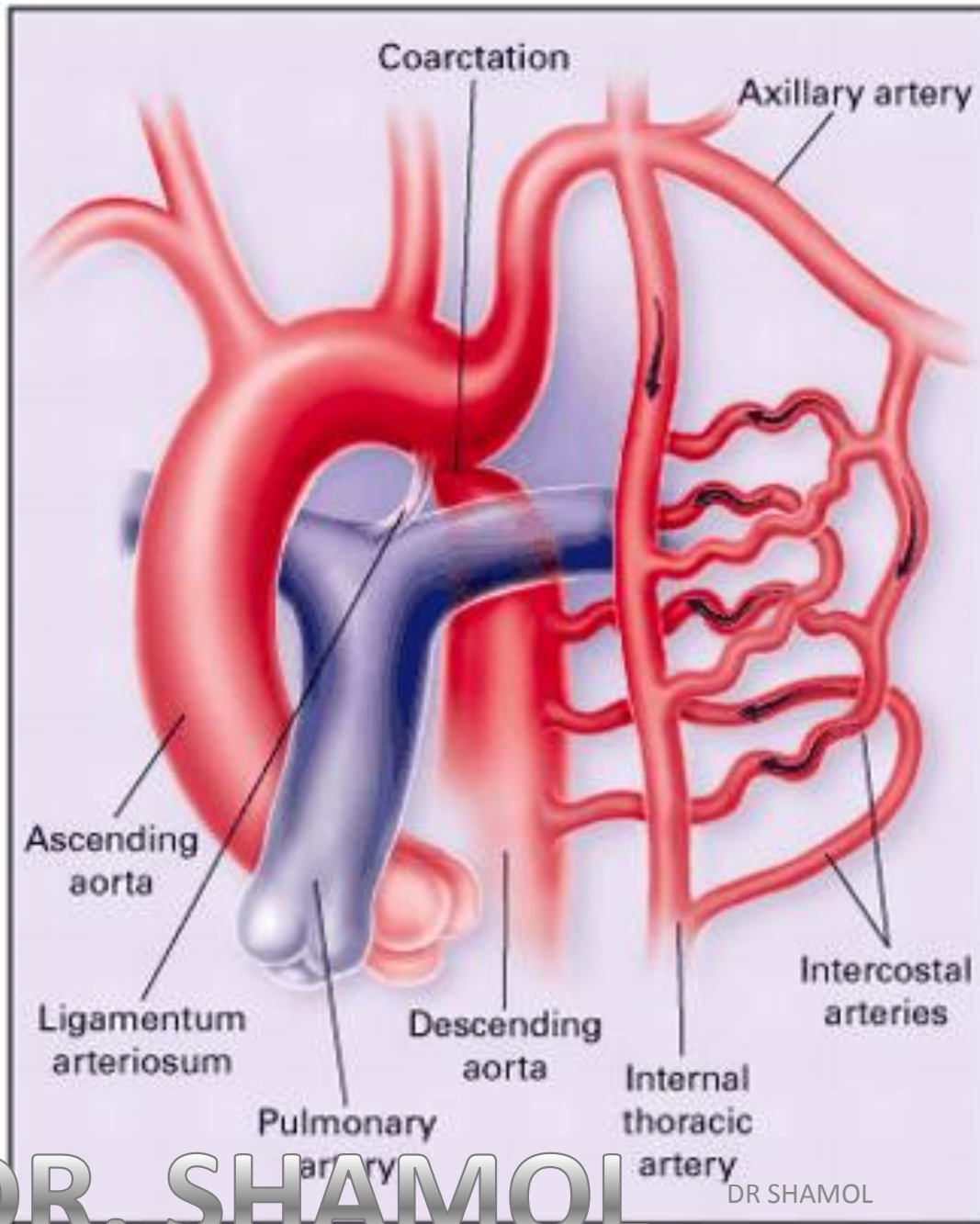
<b>What is the treatment</b>	<b>surgery</b>
	<b>Done preferably before 5 years of age</b>
	type of surgery → Surgical resection and end-to-end anastomosis
	If coarctation is extensive then prosthetic vascular graft may be done
	if surgery is not possible -- balloon dilatation and sometimes stenting.
if surgery done early in childhood	persistent hypertension can be avoided
if surgery is done during adolescence or adulthood	hypertension may persist in up to 70% cases, because of irreversible changes in arterioles
if restenosis	Balloon angioplasty and stenting

<b>Complication --HARDIS</b>	
<b>H</b>	Hypertension and its complication (LVF, CVA)
<b>A</b>	Aneurysm of aorta
<b>R</b>	Rupture at the coarctation site
<b>D</b>	Dissecting aneurysm
<b>I</b>	Infective endocarditis
<b>S</b>	Subarachnoid hemorrhage (rupture of Berry aneurysm of circle of Willis)



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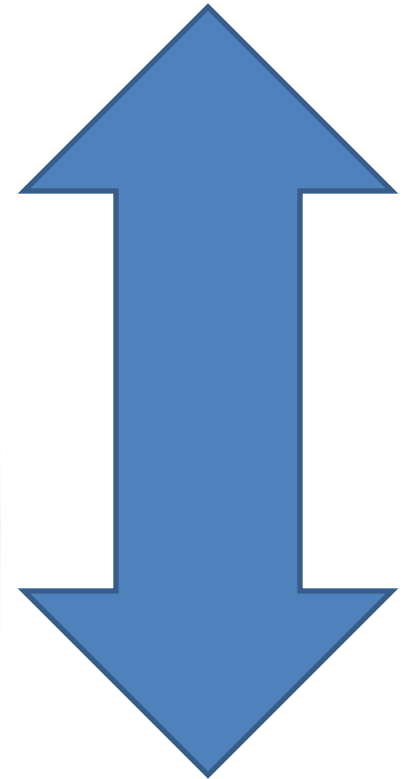
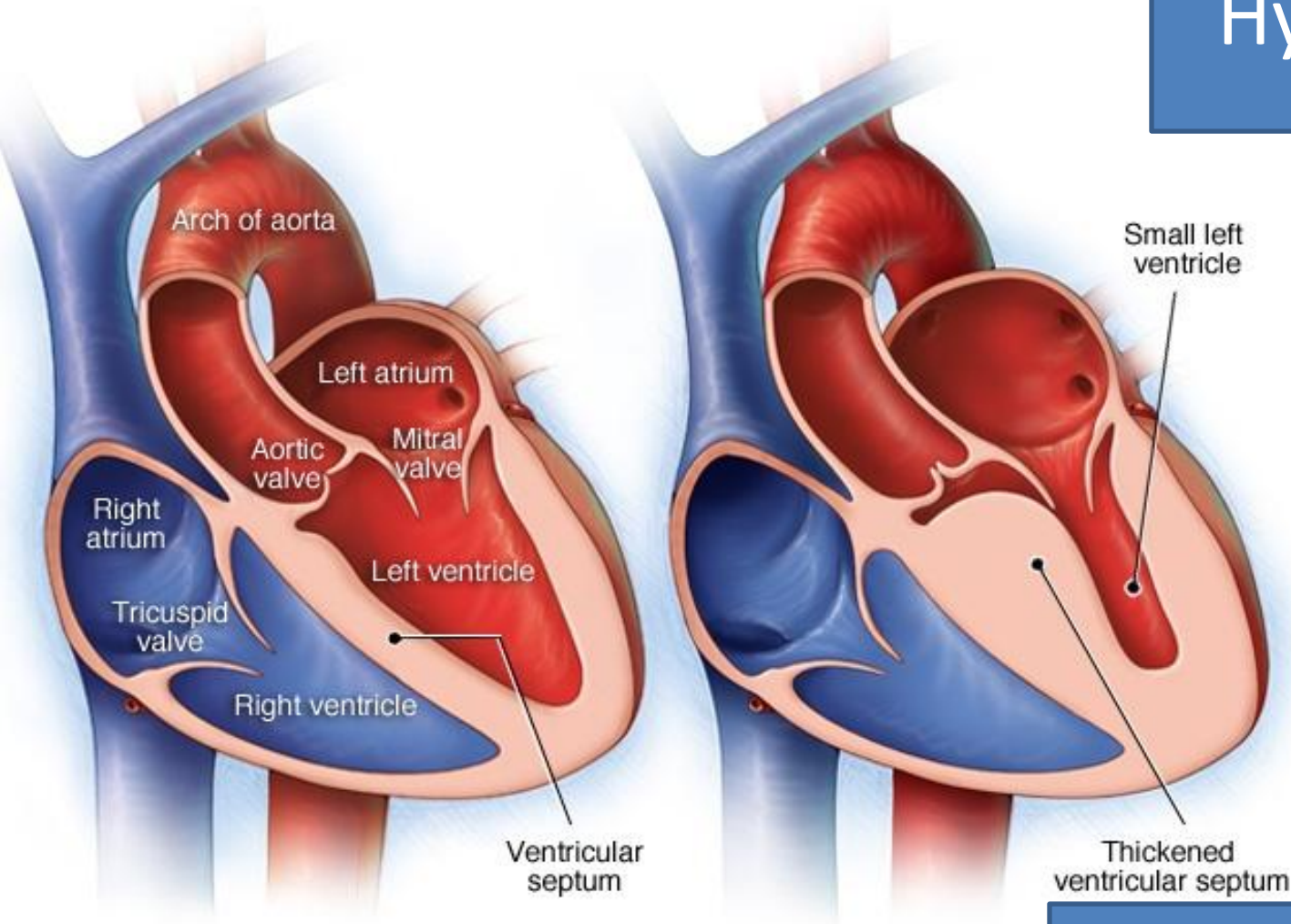
<b>What causes rib notching?</b>	Collateral flow through dilated, tortuous and pulsatile posterior intercostal arteries typically causes notching on the undersurfaces of the posterior portions of the ribs
<b>why not anterior part of rib</b>	The anterior parts of the ribs are spared because the anterior Intercostal arteries do not run in the costal grooves
<b>in which rib</b>	Notching is seldom found above the third or below the ninth rib and rarely
<b>age of appearance</b>	Appears before the age of 6 years.



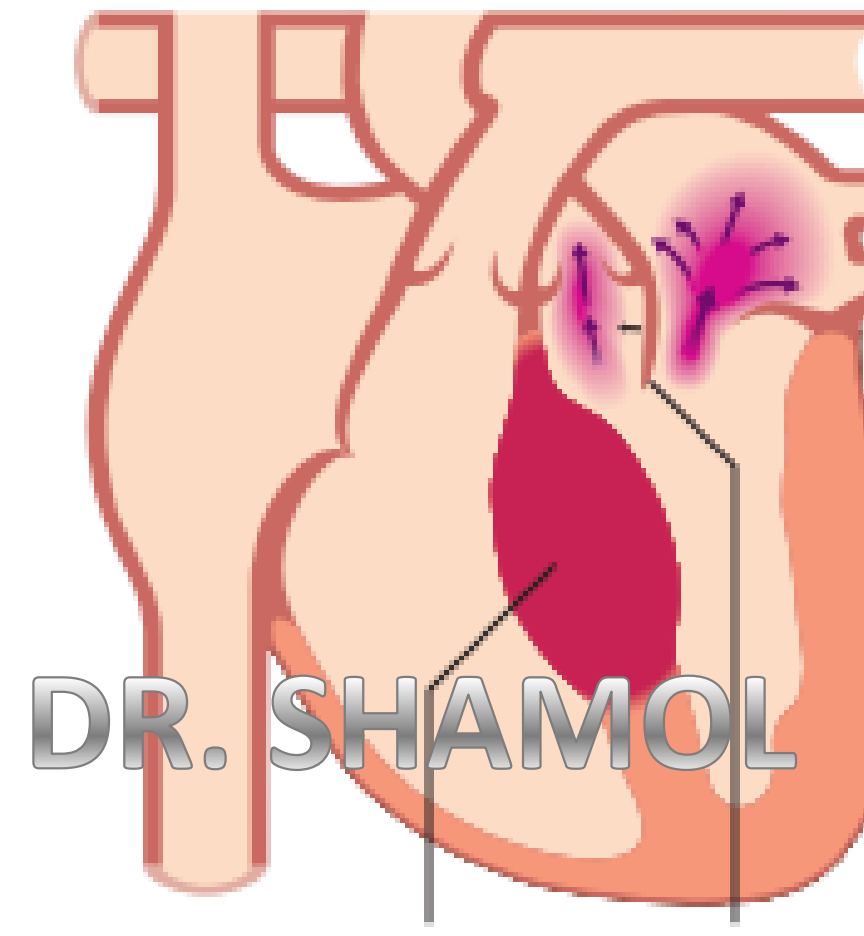
Normal

Hypertrophic cardiomyopathy

Hypertrophic



cardiomyopathy



pulse	Jerky	
BP	low systolic,	
	normal diastolic	
	narrow pulse pressure	
apex beat	Double apical impulse	
thrill	A systolic thrill may be palpable at apex from severe mitral regurgitation	
	another systolic thrill the lower left sternal border from outflow tract obstruction	
Murmur	harsh ejection systolic murmur at the left lower sternal border	
	Pansystolic murmur at the apex caused by mitral regurgitation	

**why double apical impulse**

There is a double impulse at the apex; this represents presystolic ventricular expansion from forceful atrial systole followed by a systolic left ventricular heave.

Double impulse at the apex palpable fourth heart sound due to left atrial hypertrophy

**why arterial pulse is jerky**

The arterial pulse is jerky. A sharp early rise of rapid ejection is followed by a late systolic phase.

Symptom	A	Angina on exertion
	B	Exertional Breathlessness
	C	Exertional syncope
	D	Sudden death

What is the peculiarity of ejection systolic murmur? How will differentiate from MURMUR of aortic stenosis ?

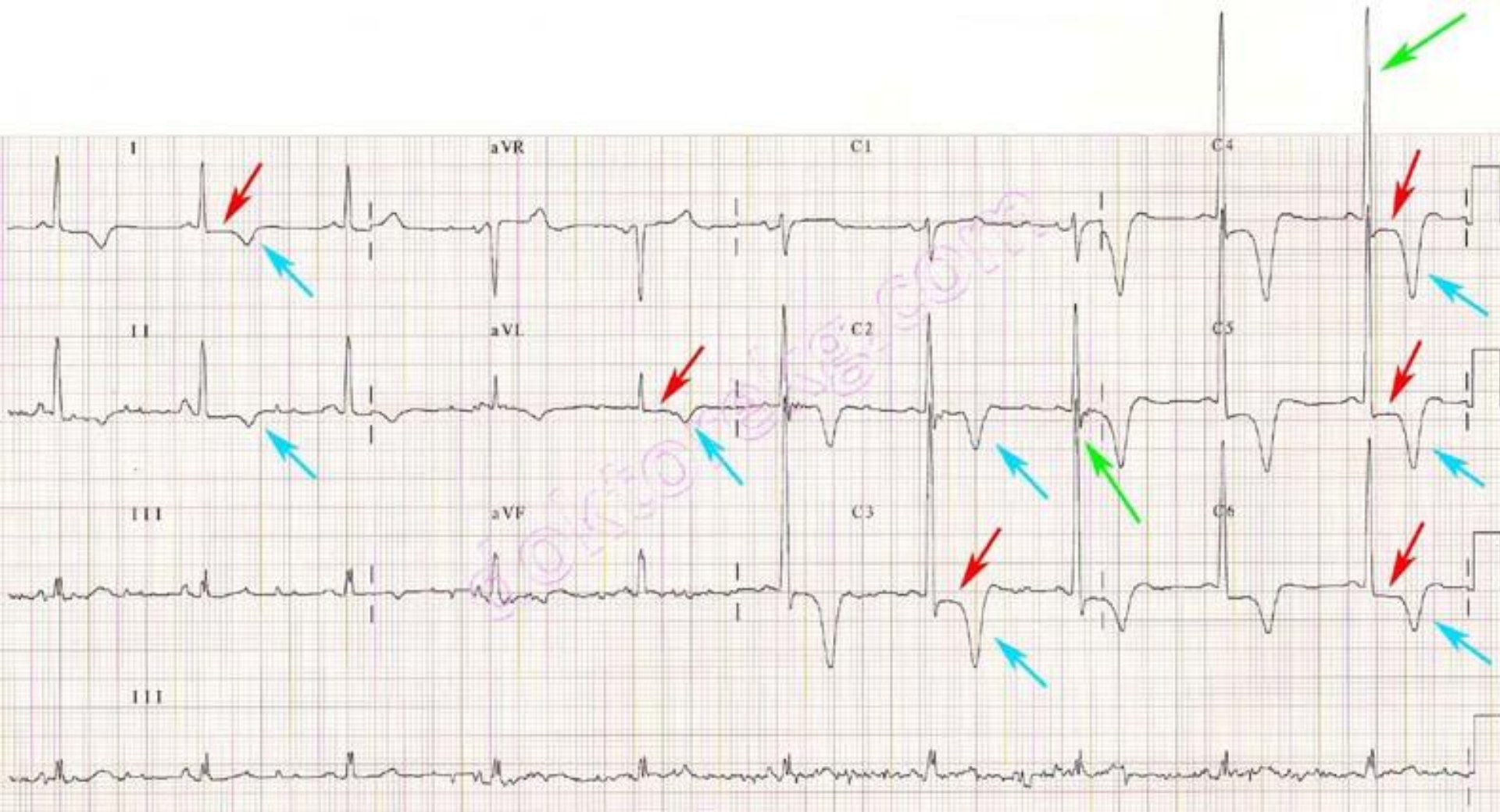
accentuated /Increased by	by standing and Valsalva manoeuvre → due to reduces LV cavity size and increases outflow tract obstruction	
decreases/softer	during squatting or sustained hand grip → due to increases LV cavity size and reduces outflow tract obstruction	
difference of AS	Valsalva manoeuvre	increases the murmur of hypertrophic cardiomyopathy
		decreases the duration of murmur of aortic stenosis

TREATMENT	there is no pharmacological treatment that is definitely known to		
pharmacological	improve prognosis		
	to relieve symptoms & prevent syncopal attack	Beta-blockers	
		Rate-limiting calcium antagonists (e.g. verapamil)	
		Disopyramide	
	if Arrhythmias	amiodarone	
	Dual-chamber pacing or DDD pacing		
intervention	relieve the out flow obstruction	partial surgical resection (myectomy) or	
		iatrogenic infarction of the basal septum (septal ablation) using a catheter delivered alcohol solution	
	if clinical risk factors for sudden death	implantable cardiac defibrillator(ICD)	
Drug should be avoided	D	Digoxin	
	D	vasodilators	ACE i
			Dihydropyridine calcium channel blockers
			Nitrates
			Alcohol (may cause vasodilatation)
	D	Diuretics	
Cardiac transplantation		needed in CHF not responding to treatment	

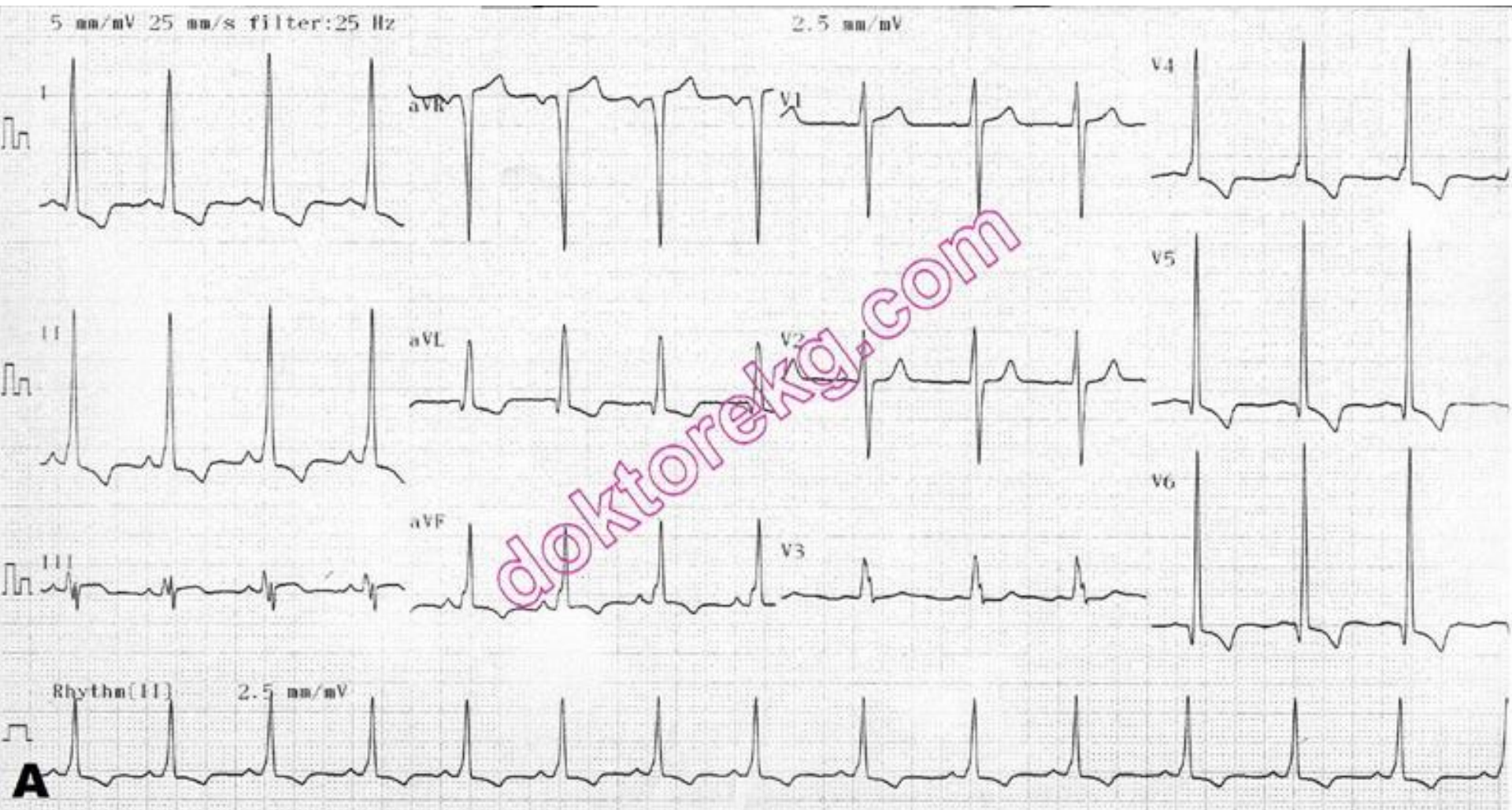
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ECG	Left ventricular hypertrophy with a wide variety of often bizarre abnormalities (70% to 80%)	in 25%case pseudo-infarct pattern, Abnormal Q waves simulating myocardial infarction→ septal hypertrophy
		deep T-wave inversion→ (particularly in anterior and inferior leads in the apical form of HCM)
		distinctive pattern of diffuse symmetric T-wave inversions across the precordium(apical HCM)
	rhythm	most patients, is normal sinus rhythm
	ambulatory monitoring demonstrate high incidence of	supraventricular tachycardia (46%)
		Premature ventricular contractions (43%)
		nonsustained ventricular tachycardia (26%)
		Atrial fibrillation25% to 30%
CXR	may be normal	
	or show evidence of left ventricular enlargement.	
Echo		
Cardiac Catheterization		
genetic testing		
Endomyocardial biopsy		



The ECG above belongs to a patient with APICAL hypertrophic cardiomyopathy. Deeply negative T waves in precordial leads , ST segment depression and voltage criteria for left ventricular hypertrophy suggested a diagnosis of apical hypertrophic cardiomyopathy. The increased R wave amplitude in right precordial leads



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RPA



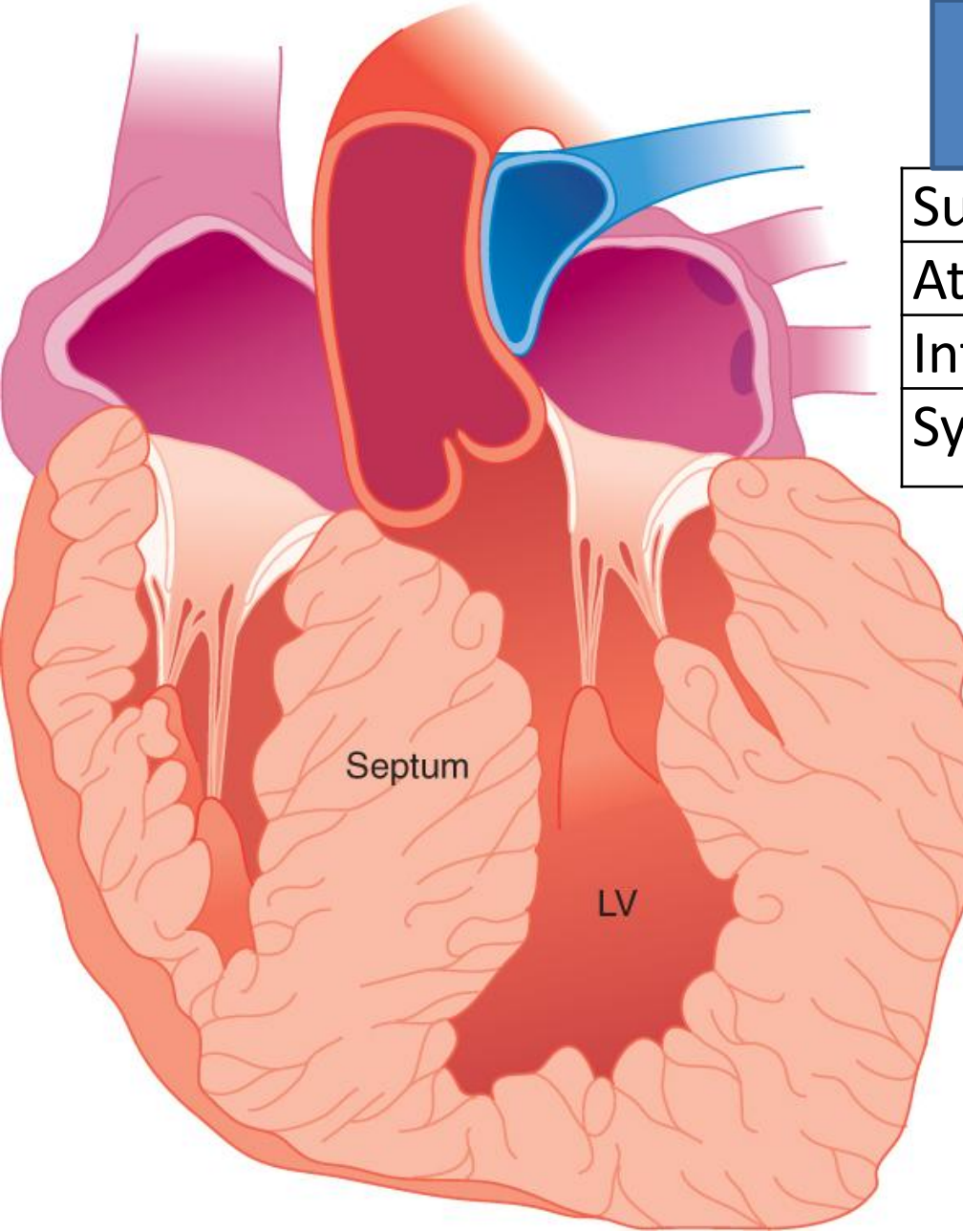
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Difference between AS and HCM		<b>AS</b>	<b>HCM</b>
	pulse	Pulse is low volume and slow rising	jerky pulse
	apex beat	heaving	Double apical impulse
	thrill	Systolic thrill in aortic area.	lower left sternal border
	2 <sup>nd</sup> heart sound	soft A2	normal
	murmur	harsh ejection systolic murmur in aortic area	harsh ejection systolic murmur at the left lower sternal border
	radiation	radiate towards neck	no radiation
	pansystolic murmur	absent	present
	Valsalva manoeuvre	Decreases the duration of murmur	Increases the murmur

deference between PS and AS		HCM	PS
	pulse	jerky pulse	normal
	apex beat	Double apical impulse	normal
	Left parasternal heave & epigastric pulsation absent	present	present
	Systolic thrill	lower left sternal border	pulmonary area
	heart sound	A2 t & P2 normal	A2 normal & P2 soft
	wide splitting of the second heart sound	absent	may present
	Ejection systolic murmur	the left lower sternal border	in pulmonary area
		no radiation	radiates to the left side of neck



## Complications

Sudden death

Atrial fibrillation

Infective endocarditis

Systemic embolization.

Risk of sudden death –CARE -, ECG & ECHO

C	A history of previous <b>cardiac arrest</b> or sustained ventricular tachycardia
A	An <b>adverse genotype</b> and/or family history sudden cardiac death (( $< 50$ years old))
R	<b>Recurrent syncope</b>
E	<b>Exercise</b> -induced hypotension/ failure to rise BP
ECG	Non-sustained ventricular tachycardia on ambulatory ECG monitoring
ECHO	Marked increase in left ventricular wall thickness ( $> 30$ mm on echo )

# Sudden Cardiac Death in HCM

